



This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

- + *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.
- + *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.
- + *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.
- + *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at <http://books.google.com/>

24503404410



L122 .B79 1907
LANE MEDICAL LIBRARY STAMFORD
STOR
The cause and prevention of beriberi /

Annex Anatomy
Old Class

~~DEC 1 8 2004~~

JAN 11 2005

~~DEC 1 9 2004~~

PLEASE DO NOT REMOVE THIS BAND

REMOTE STORAGE

1907

Please return at the circulation desk.

To renew your material call:

(650) 723-6691 ext. 3

Date due in Lane Library:

JAN 13 2005

ARY

ND

**THE CAUSE AND PREVENTION OF
BERI-BERI**

THE CAUSE AND PREVENTION OF BERI - BERI

BY
W. LEONARD BRADDON, M.B., B.S., F.R.C.S.
STATE SURGEON, NEGRI SEMBILAN, FEDERATED MALAY STATES



LONDON
REBMAN LIMITED
129 SHAFTESBURY AVENUE, W.C.

NEW YORK
REBMAN COMPANY
1123 BROADWAY

1907
S

Entered at Stationers' Hall.

All rights reserved.

Y8A98LJ 38A1

PREFACE

THIS work consists chiefly of a Report presented to the Colonial Office in May, 1904, for official permission to publish and use the material contained in which, as well as for encouragement and much opportune assistance towards the collection of information, I here offer grateful acknowledgment.

Certain facts and observations gleaned later from other authors or more recent works while the book has been at press have been interpolated here and there in the text, and others added in an appendix. I believe that no contribution of importance to the subject has been ignored, and wherever I have found facts capable of standing as evidence I have cited them and their observer. But in the literature of beri-beri there is so much that is opinion merely, so much more that is but an iteration of previous opinions—not always correctly recited—that facts of value are few and far between.

In the present work I have sought as far as possible to omit all mere opinions, and to present the reader with the evidence of *facts*.

If, therefore, it seem presumptuous to claim, as I do, that in this book a problem which has vexed medicine for centuries receives solution, that the cause and prevention of beri-beri are clearly and certainly shown, the appeal is not to the author's opinions, but to the facts recorded for justification.

I have the pleasure of expressing my thanks for much

statistical material to my colleagues in Malaya, Drs. Kerr, Leask, Mugliston, Wright, Lucy, Ellis, Fry, MacDowell, and Travers—to the latter especially I owe thanks for much stimulating criticism in consistent opposition to the views expressed in the work; to the Director-General of the Naval Medical Department; to the Principal Medical Officer, Madras Command, who, with great kindness, permitted me access to the official records as to beri-beri in the British Navy and in the Indian Army; to Drs. Clemow of Constantinople and B. G. Corney of Fiji; to Dr. Nightingale, lately, and Dr. Night, now, of Bangkok, Dr. Arnott of St. Helena, Dr. MacDougall of Christmas Island, Lieut.-Col. C. G. D. Mosse, and Major W. Buchanan, for valuable notes personally supplied; to the Surgeon-General with the Government of Madras; to the Surgeon-General and the Chief Surgeon of the Philippine Division, U.S.A., and to Dr. Hodenpijl, Inspector of the Medical Service, R.D.N., for information officially given.

Last, I owe great obligation to my friends, Dr. W. E. Boycott and Canon W. M. Roberts, who have relieved me of much of the burden of seeing the book through the press—the former having put me under especial obligation in the correction of references, the latter in the revision of proofs and compilation of the index.

W. L. B.

SEREMBAN.

CONTENTS

SECTION I

GENERAL CONSIDERATIONS

	PAGES
PREVALENCE IN THE MALAY PENINSULA - - - -	I
MORTALITY IN HOSPITALS - - - - -	2-4
OUTSIDE HOSPITALS - - - - -	4
ECONOMIC LOSS FROM THE DISEASE - - - -	5
CLINICAL CHARACTERISTICS :	
VARIABLE COURSE OF THE DISEASE - - - -	6
DISTINCTIVE TYPES CLASSIFIED - - - -	7
PRODUCTION OF 'CHRONIC' PARALYTICS - - - -	8
EFFECT OF CHANGE OF ENVIRONMENT - - - -	9
SYMPTOMS ALWAYS ASCENDING AND SYMMETRICAL - - - -	10
HYPERMYOTONUS - - - - -	10
ANALOGY WITH 'LANDRY'S PARALYSIS' - - - -	11
WITH ACUTE ANTERIOR POLIOMYELITIS - - - -	12
WITH ALCOHOLIC AND ARSENICAL NEURITIS - - - -	12
OEDEMA IN INITIAL STAGES OCCASIONALLY ASYMMETRICAL - - - -	13
SYMPTOMS INDICATING CENTRAL EFFECTS OF THE POISON - - - -	14
STOMACH INFLAMMATION FREQUENT - - - -	15-17
RESEMBLANCE TO OTHER SPINAL DISORDERS - - - -	18
VARIOUS DISEASES MISTAKEN FOR BERI-BERI - - - -	19-20
SALIENT EPIDEMIC FEATURES - - - - -	21
BERI-BERI BELONGS TO THE DISEASES WHICH ARE DUE TO GENERAL POISONS - - - - -	22
APPARENT INCOMPATIBILITY BETWEEN ITS MOST MARKED CHAR- ACTERS - - - - -	23
THEORIES AS TO THE ETIOLOGY OF BERI-BERI :	
BACTERIAL—DISPROVED BY AUTHOR'S EXPERIMENTS - - - -	24-25
INFECTIOUS—WRIGHT'S AND DURHAM'S EXPERIMENTS CRITI- CISED - - - - -	26-29
DISPROVED BY TRAVERS' EXPERIMENT - - - -	30-31

	PAGES
MIASMATIC—DOES NOT ACCOUNT FOR THE GREAT MAJORITY OF CASES - - - - -	32-35
INCOMPATIBLE WITH THE INEQUALITY IN THE INCIDENCE OF THE DISEASE - - - - -	35-37
ONLY POSSIBLE ORIGIN REMAINS— <i>i.e.</i> , OF A POISONING THROUGH FOOD - - - - -	38

SECTION II

**A THEORY OF GRAIN INTOXICATION AS THE CAUSE
OF BERI-BERI**

TOXIC EFFECT OF FOOD AS A CAUSE OF THE DISEASE :	
NOT THROUGH A DIET PHYSIOLOGICALLY INCORRECT -	39
NOR THROUGH FOOD BECOMING DAMAGED OR DECOMPOSED -	40
BUT MAINLY THROUGH THE CONSUMPTION OF RICE -	41
RICE NOT, HOWEVER, THE SOLE CAUSE, WHICH MAY BE ANY CEREAL - - - - -	42-43
NATURE OF THE CONNECTION BETWEEN BERI-BERI AND RICE AS A	
STAPLE FOOD - - - - -	44
NO BERI-BERI WHERE THERE IS NOT A RICE DIET -	45
ACTION OF GRAIN PARASITES - - - - -	45-46
RESEMBLANCE OF BERI-BERI TO OTHER DISEASES DUE TO GRAIN- POISONING - - - - -	46-47
DEDUCTIONS FROM THE RICE POISON THEORY TO BE VERIFIED IN THE COURSE OF THE WORK - - - - -	48-57

SECTION III

THE LATENT OR INCUBATION PERIOD OF BERI-BERI

UNIFORMITY OF PERIOD FOR ACUTE INFECTIOUS DISORDERS -	58
DIFFICULTY OF DETERMINING THIS PERIOD IN BERI-BERI -	59
EVIDENCE AS TO AVERAGE PERIOD OF INCUBATION :	
FROM INDIVIDUAL OBSERVATIONS - - - - -	60-66
FROM STATISTICS OF PUBLIC INSTITUTIONS - - - - -	67-85
GENERAL INFERENCE FROM ABOVE EVIDENCE - - - - -	85
CONFIRMED FROM OTHER SOURCES - - - - -	86
THESE RESULTS INCOMPATIBLE WITH THE INFECTION THEORY, BUT CONSISTENT WITH THE RICE-POISON THEORY -	87
NON-RECOGNITION OF THE POSSIBLE EXTENT OF THE LATENT PERIOD A SOURCE OF ERROR - - - - -	88-90

CONTENTS

ix

SECTION IV

PROOF OF DEDUCTIONS MADE FROM THE THEORY OF GRAIN INTOXICATION IN THE CASE OF RICE

	PAGES
(A) BERI-BERI MUST SHOW A DIRECT RELATION TO RICE-SUPPLY	91
SUBSIDIARY PROPOSITIONS :	
1. IN EPIDEMIES OF BERI-BERI AND IN PLACES WHERE IT IS ENDEMIC, RICE-EATERS ONLY ARE ATTACKED ; THOSE WHO EAT NO RICE ESCAPE - - - -	92
EVIDENCE OF THE ABOVE PROPOSITION :	
SUPPLIED BY SHIPS - - - -	92-100
FROM EPIDEMIES ON LAND - - - -	100-117
IN PARTICULAR FROM THE MALAY GAOLS AND ASYLUMS	117-125
FROM DIFFERENCE OF DIET DUE TO ETHNOLOGICAL CHARACTERISTICS - - - -	125-135

SECTION IV (continued)

2. AMONG RICE-EATERS DURING EPIDEMIES, AND IN PLACES WHERE IT IS ENDEMIC, ITS INCIDENCE VARIES WITH THE SORT OF RICE EATEN :	
PRELIMINARY DISCUSSION OF THE STRUCTURE AND COMPOSITION OF RICE-GRAINS - - - -	137-141
MODES OF PREPARATION OF RICE-GRAINS - - - -	141-148
TOXICITY DEPENDS ON THE MODE OF PREPARATION - - - -	148-150
CONNECTION BETWEEN BERI-BERI AND 'UNCURED' RICE PROVED BY THE DIFFERENT INCIDENCE UPON THE CHINESE AND TAMIL IMMIGRANTS INTO BRITISH MALAYA - - - -	150-154
TAMILS USING 'CURED' RICE EXCLUSIVELY ARE BERI-BERI FREE, WHILST AMONG CHINESE USING 'UNCURED' RICE BERI-BERI IS EVERYWHERE PREVALENT - - - -	154-171
WHEN THE CONDITIONS ARE REVERSED THE INCIDENCE IS REVERSED - - - -	171-173
PROVED ALSO BY THE INCIDENCE UPON THE MALAYS, AMONGST WHOM IT IS COMPARATIVELY A NEW DISEASE - - - -	173-192
CONCLUSION CERTAIN THAT IN MALAYS THE INCIDENCE OF BERI-BERI DEPENDS ON SORT OF RICE CONSUMED - - - -	193-198
FURTHER EVIDENCE IN SUPPORT OF THIS PROPOSITION :	
FROM MADAGASCAR - - - -	198-199
FROM JAVA - - - -	200-206
FROM SIAM - - - -	207-209
FROM INDIA - - - -	210-212

SECTION IV (*continued*)

(B) THE INCIDENCE AND CONSEQUENT MORTALITY OF BERI-BERI SHOULD VARY WITH THE QUANTITY, ABSOLUTE OR RELATIVE, OF RICE EATEN :

EVIDENCE FROM CHINESE IMMIGRANTS INTO BRITISH MALAYA	213-223
FROM MANILA - - - - -	223-224
EVIDENCE FROM JAPANESE ARMY, FLEET, AND PRISONS -	224-233
FROM THE HISTORY OF VARIOUS EPIDEMIES -	233-240
FROM THE DUTCH EAST INDIAN NAVY -	240-244
FROM THE APPEARANCE OF BERI-BERI ON ITALIAN AND ENGLISH WARSHIPS - - - - -	245
FROM THE DUTCH EAST INDIAN ARMY -	246-249
FROM THE SINGAPORE GAOL - - - - -	249-251
FROM THE SELANGOR PRISON - - - - -	252-254
FROM THE SINGAPORE LUNATIC ASYLUM - - - - -	254
FROM JAPANESE COOLIES IN FIJI - - - - -	254-255
FROM THE DUTCH ARMY AND FLEET - - - - -	255-256
EFFECT OF FAT IN DIET - - - - -	257-260
EFFECT OF IMPROVED DIETARY - - - - -	260-262
BERI-BERI AMONGST PRISONERS IN BATAVIA - - - - -	262-266
SPECIAL INCIDENCE ON RECRUITS - - - - -	266-271
IN SIAM - - - - -	271-272
IN THE CONGO STATE - - - - -	272-274
SURGICAL AND PUERPERAL - - - - -	275-278

SECTION IV (*continued*)

(C) AS DEPENDENT MERELY UPON THE SORT OF RICE EATEN, BERI-BERI WILL PERSIST AS LONG AS THE SAME RICE IS USED, AND DISAPPEAR WHEN THIS IS DISCONTINUED:

EVIDENCE FROM THE DUTCH EAST INDIAN FLEET -	280-282
FROM AN EPIDEMY IN JELEBU - - - - -	282-284
FALLACIOUS INFERENCES FROM CHANGE OF LOCALITY -	284-287
TRAVERS' AND WRIGHT'S EXPERIMENTS IN PUDOH GAOL CRITICISED - - - - -	287-293
EVIDENCE THAT THE COURSE OF THE DISEASE IS CHECKED MERELY UPON CHANGING THE SORT OF RICE EATEN -	293-307
EXPERIMENTAL DETERMINATIONS OF COMPARATIVE TOXICITY OF DIFFERENT RICES - - - - -	307-319
RECAPITULATION - - - - -	319-321
THE AUTHOR'S THEORY EXPLAINS MANY EXTRAORDINARY AND CONFLICTING FEATURES OF THE DISEASE - - - - -	321-325

SECTION V

PERIODIC MOVEMENTS OF BERI-BERI

SEASONAL :

NOT DEPENDENT ON CLIMATE - - - - -	326-327
SPECIAL FACTORS IN PERIODICITY - - - - -	327-335

CONTENTS

xi

PAGES

MULTI-ANNUAL :

INDEPENDENT OF METEOROLOGICAL FACTORS	-	-	335-339
BUT SIMULTANEOUS IN DIFFERENT STATIONS	-	-	339-346
INFERENCES FROM ABOVE AS TO ETIOLOGY OF BERI-BERI	-	-	346-349

SECTION VI

BERI-BERI IN ANIMALS

IN HORSES	-	-	-	-	-	-	350-355
IN MONKEYS	-	-	-	-	-	-	355-356
IN FOWLS—EIJKMAN'S AND SAKAKI'S EXPERIMENTS	-	-	-	-	-	-	356-365
EFFECTS FOLLOWING THE USE OF 'PADI'	-	-	-	-	-	-	365-367

SECTION VII

NATURE OF THE TOXIC AGENT IN RICE WHICH PRODUCES BERI-BERI

DISCUSSION OF THE QUESTION WHETHER BERI-BERI IS DUE, NOT TO THE PRESENCE OF A POISON, BUT THE OCCASIONAL ABSENCE OF SOMETHING NORMALLY PRESENT IN RICE	-	-	-	-	-	-	368-370
INFERENCES FROM EVIDENCE ADDUCED FROM PREVIOUS SECTIONS AS TO CONDITIONS UNDER WHICH RICE BECOMES TOXIC :							
1. STALE (WHITE) RICE OCCASIONALLY CONTAINS A POISON WHICH PRODUCES BERI-BERI	-	-	-	-	-	-	370
2. THE AGENT WHICH PRODUCES THIS POISON IS PECULIAR TO THAT GRAIN	-	-	-	-	-	-	371
3. THE POISON IS NOT ENDOGENIC IN NORMAL FRESH SEEDS, BUT IS ADVENTITIOUS	-	-	-	-	-	-	371
4. THE PERICARP OF RICE, WHEN FRESH, CONTAINS LITTLE OR NO POISON, POISON ARISING ONLY AFTER DECORTICATION	-	-	-	-	-	-	372
5. THEREFORE THE FORMATION OF POISON IS DUE TO THE ACTION OF A SPECIFIC AGENT ON THE DEAD SEED	-	-	-	-	-	-	372-374
6. THE AGENT MUST BE SOME FERMENT OR PARASITE OR EPIPHYTE PECULIAR TO 'PADI'	-	-	-	-	-	-	374-376
EXACT NATURE OF TOXIN AND PROCESS OF FORMATION AS YET UNKNOWN	-	-	-	-	-	-	376-377
PHYSICAL PROPERTIES OF TOXIN	-	-	-	-	-	-	377-378
TOXICOLOGICAL AFFINITIES	-	-	-	-	-	-	378-385
7. FROM THE ABOVE IT WOULD FOLLOW THAT THE BERI-BERIC POISON IS A STABLE AND NON-VOLATILE ALKALOID RESEMBLING ATROPINE AND MUSCARINE IN ITS EFFECTS	-	-	-	-	-	-	385

	PAGES
8. THE FORMATION OF POISON IN STALE RICE IS PROBABLY DUE NEITHER TO FERMENTATION NOR TO BACTERIA, BUT TO THE GROWTH IN IT OF A SPECIAL FUNGUS -	385-390
9. THE FUNGUS OF RICE WHICH PRODUCES BERI-BERI IS PROBABLY A SURFACE PARASITE OR EPIPHYTE AFFECT- ING THE SEED SAPROPHYTICALLY AFTER DECORTICA- TION - - - - -	390-392
10. THE SPECIFIC FUNGUS OF BERI-BERIC RICE IS, LIKE THAT OF TOXIC RYE AND LOLIUM, PROBABLY A PARA- SITE AFFECTING THE SURFACE OF THE SEED -	392-396
THIS HYPOTHESIS HELPS TO EXPLAIN MANY DIFFICULTIES IN THE NATURAL HISTORY OF THE DISEASE -	396-399
AND THE POSSIBILITY OF OTHER CEREALS BECOMING A CAUSE OF BERI-BERI - - - - -	399-400

SECTION VIII

OBJECTIONS TO PREVIOUS RICE THEORIES

THE VALIDITY OF THESE OBJECTIONS RESTS UPON THE PROOF
OF THE FOLLOWING STATEMENTS:

1. THAT IN CASES WHERE RICE SHOULD HAVE PRODUCED
BERI-BERI, BUT FAILED TO DO SO, IT WAS (a) OF SUCH
A SORT AS COULD BE TOXIC; (b) WAS TAKEN IN SUFFI-
CIENT QUANTITY; AND (c) FOR A SUFFICIENTLY LONG
PERIOD
2. THAT IN CASES WHERE BERI-BERI HAS APPEARED, BUT
THE DIRECT USE OF RICE CANNOT BE TRACED, IT HAS
BEEN USED IN A MASKED FORM—*e.g.*, AS ARROWROOT
OR GLUCOSE—OR THE FOOD IN USE HAS BEEN EX-
POSED TO POSSIBLE CONTAMINATION BY STALE RICE
3. THAT THE DISORDER DIAGNOSED AS BERI-BERI WAS THE
TOXIC DISEASE - - - - - 401-402

DISCUSSION OF VARIOUS OBJECTIONS THAT IGNORE ONE OR
OTHER OF THESE CONDITIONS - - - - - 403-423

BERI-BERI IN TEMPERATE LATITUDES:

- A PERIPHERAL NEURITIS NOT IDENTICAL WITH, BUT POS-
SIBLY A MODIFIED FORM OF, TRUE BERI-BERI, AND THE
RESULT OF THE SAME AGENT—THEREFORE A GRAIN
INTOXICATION - - - - - 423
- VARIOUS CAUSES OF PERIPHERAL NEURITIS - - - - - 424-426
- DISCUSSION OF THREE RECENT EPIDEMIES - - - - - 426-441
- INDIRECT MODES OF RICE CONSUMPTION:
- ADULTERATION OF FLOUR - - - - - 441-443
- ADULTERATION OF GLUCOSE - - - - - 443-447
- RICE TOXIN IN OTHER CEREALS - - - - - 447-448
- SPECIAL LIABILITY OF THE INSANE TO BERI-BERI - - - - - 449-451

CONTENTS

xiii

SHIP BERI-BERI :	PAGES
IDENTITY WITH TRUE BERI-BERI DOUBTFUL - -	452-457
DISCUSSION OF SPECIAL REPORTS OF SHIP EPIDEMIES -	457-464
VARIOUS THEORIES ADVANCED - - -	464-470
SUMMARY AND CONCLUSIONS FROM FOREGOING EVIDENCE	471-472
NOTE ON THE RELATION BETWEEN BERI-BERI AND ARSENIC :	
EPIDEMY OF NEURITIS AMONGST BEER-DRINKERS -	473-478

SECTION IX

THE PREVENTION OF BERI-BERI - - -	479
-----------------------------------	-----

APPENDIX I

NOTE ON TRAVERS' SELANGOR GAOLS EXPERIMENT AS TO THE	
EFFECT OF DIET ON BERI-BERI - - -	481-501
DIETS AND BERI-BERI IN THE SINGAPORE AND SELANGOR GAOLS	501-512

APPENDIX II

TABLES SHOWING ADMISSION OF CHINESE PATIENTS FOR ALL	
CAUSES, FOR BERI-BERI, AND THE MORTALITY FROM	
BERI-BERI IN THIRTY-ONE HOSPITALS IN THE STRAITS	
SETTLEMENTS AND NATIVE MALAY STATES BETWEEN 1881	
AND 1902 - - - - -	513-521
LITERATURE - - - - -	522-535
INDEX - - - - -	536-544

THE CAUSE AND PREVENTION OF BERI-BERI

SECTION I

GENERAL CONSIDERATIONS

PREVALENCE; MORTALITY; CLINICAL, ANATOMICAL, AND EPIDEMIOLOGICAL CHARACTERISTICS; THEORIES OF INFECTION AND MIASM DISPROVED; OLDER FOOD-INTOXICATION THEORIES CONSIDERED

Prevalence in British Malaya.—In the colony of the Straits Settlements and adjacent Native States of the Malay Peninsula, an area about equal to that of England, but with a total population of only a million and a quarter, over 150,000 cases of beri-beri have been treated, and 30,000 have died, during the last two decades, in Government hospitals and infirmaries alone.¹

Rate of Incidence on Chinese.—Among the Chinese immigrants into this region, whom it almost exclusively affects, it may be reckoned that of every 1,000 living, 120 suffer from it in some degree, 80 are severely attacked, and 16 die of it annually.

Assessed at a money value, the losses directly entailed by it upon employers amount to some million of dollars every year, while the charges upon the Government caused by the maintenance of the sick equal a tenth or more of that sum.

The evils of beri-beri include not only the conditions it directly produces. A large number of its victims are permanently disabled; many become vagrants; all afterwards, more liable to disease, help to swell the hospital population.

Not only so, but the condition which produces beri-beri renders those exposed to it more prone to other disorders than the

¹ About one-third of all deaths of Chinese took place in Government hospitals. The total deaths from beri-beri during the period, therefore, would be some 100,000!

2 THE CAUSE AND PREVENTION OF BERI-BERI

healthy, even before an actual attack of it has become declared. The evidence of this is, that wherever beri-beri is most prevalent, the general sickness rate from other disorders also is usually much increased.

Total Mortality due to Beri-beri.—It is likely that, when to the direct attacks of beri-beri the illnesses indirectly entailed by it have been added, the total mortality for which the disease is responsible will be more nearly four- than two-fifths of the whole death-rate, or 32 per mille.

Under a burden of this kind no ordinary population could long maintain itself. That in the region referred to the Chinese are not extinguished is due to the fact that they are not a fixed population. Few of those who survive stay in the country more than five or six years continuously, while those who die are replaced by fresh immigrants, the tide of whom has doubled the numbers of their race in British Malaya within the last decade.

General Incidence as gauged by Hospitals.—The taking of a census in 1891 and 1901 enables an accurate statement to be presented of the proportion of Chinese inhabitants of British Malaya treated for, and dying of beri-beri in *hospitals* in those years.

They appear as follows :

Proportion of Chinese Adult Male Population treated for, or dying of Beri-beri in Government Hospitals annually, per 10,000 Living.

		Singapore.	Penang.	Province Wellesley.	Malacca.	Perak.	Selangor.	Sungei-Ujong.	Whole Population.
1891	Cases	78	61	10	475	175	360	392	221
	Deaths	24	15	2	90	12	66	46	36
								Nagri-Sembilan.	
1901	Cases	111	78	15	212	359	219	358	193
	Deaths	39	23	5	47	56	33	14	31

The variations in the prevalence and severity of beri-beri in districts which are geographically close together, and differ little at any time in their physical conditions, are extraordinary ; but that they are not dependent upon artificial factors, such as extent of hospital accommodation, accessibility of relief to patients etc., is shown by the fact that, whilst the admissions to hospital in the same districts for diseases other than beri-beri have increased in every case where beri-beri has increased, they have also in some instances done so where beri-beri has declined.

The following figures represent the

Rates of Admission to Hospital for all Diseases exclusive of Beri-beri, per 10,000 of living Chinese Male Adults annually.

Year.	Penang.	Province Wellesley.	Malacca.	Perak.	Selangor.	Negri-Sembilan.	Average of whole Area.
1891	713	95	1,275	257	694	324	273
1901	757	268	2,247	989	932	532	560

The table illustrates the statement made above that diseases other than beri-beri are affected by its mutations.

The next table shows the

Proportion of Cases of Beri-beri in every 1,000 Cases of all Disease (Chinese only) admitted.

Year.	Penang.	Province Wellesley.	Malacca.	Perak.	Selangor.	Negri-Sembilan.	Average of whole Area.
1891	61	10	475	175	360	392	222
1901	78	15	212	359	219	358	193

One-fifth of all Sickness Beri-beri.—More than one-fifth of all cases treated in the hospitals are beri-beri.

The case-mortality varies enormously in epidemics, and may be anything between the recovery of all or the death of all those attacked.

The writer has chronicled 100 consecutive cases admitted to hospital among whom there was not a death. He has known small groups of persons, every one of whom was attacked and perished. At Port Dickson Hospital in 1902 there were 192 cases admitted, two only of whom died, both from inter-current disorders.

Of 100 coolies imported in 1889 to labour at a particular mine, 90 were attacked within a year and died.

Travers,¹ the State surgeon of Selangor, reports 218 cases treated consecutively at one district hospital (Kajang, in 1901), with only 2 deaths; the same year at the lunatic asylum in the same State the case-mortality was 71 per cent. At the Tan Tock Seng Hospital in Singapore the death-rate has been as much as 80 per cent. Of 44,664 cases treated between 1883 and 1902 in the Selangor hospitals, the rate of mortality was 187 per 1,000.

¹ E. A. O. Travers, *Selangor Government Gazette*, 1902.

4 THE CAUSE AND PREVENTION OF BERI-BERI

The maximum in this series was 43 per cent. in the first year, the minimum 12 per cent. in the last.

The case-mortalities of different districts for 1891 and 1901 are compared in the subjoined table :

Showing Case-mortality per 1,000 Admissions for Beri-beri.

Year.	Singapore.	Penang Province.	Wellesley.	Malacca.	Perak.	Selangor.	Negri-Sembilan.	Average.
1891	?	217	200	123	129	126	126	165
1901	433	354	393	288	172	107	79	197

Thus, one out of every six cases brought into hospital dies.

In Perak, in 1899, 20.8 per cent. of all *deaths* among Chinese in hospital were from beri-beri ; in Selangor, in 1901, the proportion was one-quarter ; in Negri-Sembilan, the same year, it was one-fifth.

The average for all stations was one-fifth.

General Mortality 40 per 1,000.—The gross mortality from all causes among Chinese also varies naturally in different districts. The average for the whole region of the Straits Settlements and Native States is over 40 per 1,000 Chinese living, per annum.

Absolute Beri-beri Incidence and Mortality.—On the above data, and assuming the disease to be not more fatal outside than it is in the hospitals, it may be calculated that there occur annually among the Chinese male adult population—which in 1901 numbered 474,282 persons—34,971 cases of beri-beri, with 6,994 deaths, or a mortality of 8 and a case-incidence of 40 per 1,000.

Beri-beri Case-mortality outside Hospitals.—There can be little doubt, however, that the mortality is much more severe among the sick who are not brought to hospital. Careful investigation of the causes assigned for deaths at registration during three years in Negri-Sembilan showed that the proportion of deaths from beri-beri was relatively twice as great outside as in the hospitals. Two-fifths of all the deaths registered were assignable to it, and this at a time when the actual case-mortality from the disease was everywhere very low compared with previous years—79 per 1,000. On this basis a mortality of 16 per 1,000 yearly must be ascribed to it, the case-incidence being the same.¹

¹ At Kuala Lumpur, Selangor, the medical officer of health's reports to the Sanitary Board (monthly) show that the average mortality *from all causes for all races* is about 60 per 1,000 per annum. *One-third* of the deaths are from *beri-beri*. About the same mortality obtains in Singapore.

These rates represent only cases of the same degree of gravity as those brought into hospital. Were every case, however mild, included, the number of persons actually attacked would probably amount to two or three times this proportion, the case-mortality appearing correspondingly lower.

Direct Cost of Beri-beri to Federated Malay States Government.—Seeing that every beri-beric admitted to hospital spends a month there at least, the cost to the local government of this single disease may be calculated as not less than £10,000 annually in direct charges. Indirectly it costs probably much more.

Total Indirect Losses from Beri-beri in Federated Malay States.—The economic loss to the country at large is more difficult to assess. The Government suffers to some degree in the loss of producers of revenue, but these are replaceable. In whatever quarter the loss falls, the money value represented by the death of 7,000 labourers, and the incapacitation of five times that number for more or less prolonged periods—from weeks to years—must be very large, and may be estimated merely in wages which could have been earned as amounting to over a million dollars annually.

Origin Long Mysterious.—Long as it has been known, beri-beri has so completely baffled inquirers into its origin that it has bidden fair to become almost an accepted mystery to medicine.

Within recent years, however, the opening up and development of the region already mentioned, in which the disease is 'endemic,' has raised up evidence of a kind not accessible to earlier observers in a body of facts no less authoritative than abundant, permitting clear conclusions as to its origin to be obtained.

The solution of the problem which the overwhelming evidence of these facts affords is that beri-beri is a 'grain-intoxication,' the result of consuming a poison contained usually in rice, and in substances adulterated with rice and its derivatives, but sometimes in other cereals, and possibly at times in maize, sago, tapioca, and even in meat.

Definition.—Upon the well-ploughed fields of the clinical and anatomical features of beri-beri, save in so far as facts to be gleaned bear upon its etiology, it will not be necessary to dwell in this treatise. But it may be well at the beginning to say that the disease to which the observations apply is that which Manson has defined, in an article admirably luminous and concise, in the *Lancet*, November 23, 1901, as 'a form of peripheral neuritis, which occurs endemically and epidemically, and is especially characterized by . . . proneness to œdema and to implication of

6 THE CAUSE AND PREVENTION OF BERI-BERI

the neuro-muscular system of the central organ of circulation ; by complete absence of trophic skin-lesions, of paresis of the muscles of the head and neck, of marked implication of the organs of sight, hearing, taste, and smell, and of the mental faculties ; . . . there are troubles of locomotion, paræsthesiæ of various descriptions, especially in the lower extremities ; marked hyperæsthesia of the muscles involved, with subsequent atrophy,' etc.

Symptoms Emphasized.—Paralysis of the diaphragm especially is extremely common, but not always complete, nor always fatal. The presence of patches of anæsthesia, which are at first of small extent and 'wandering'—*i.e.*, variable in position and degree from day to day—is, when it can be determined—a matter not always easy in natives—a sign perhaps pathognomonic.

The absence of perception of light cotton-wool touches over areas corresponding to the third and second sacral segments of the cord is one of the earliest signs of incipient beri-beri. Languor, lassitude, general itching, dyspepsia, fatigue on slight exertion, tachycardia, are first among its symptoms.

Course Extremely Variable.—The course of the disease is variable, even extremely so. A few slight but unmistakable signs of it may occur, and then wholly subside, never to return.

The vast majority of cases of beri-beri consists, it is important to bear in mind, of but such slight attacks. They form the numerous class of patients in hospitals who are discharged, professing to find themselves well, within a few days after admission.

How common such cases are is probably seldom realized. They, in fact, are not noticed if not specially sought.

The writer, in February, 1902, examined nineteen Malay police in barracks at Seremban, being the part of the local force off duty at the time. None of these men professed themselves ill, or admitted that they felt ill ; yet in seventeen there were definite signs of beri-beri—anæsthesia in small patches on back of thigh, about inner malleoli, dorsum of toes, or inner side of calves ; enlarged tactile areas over spaces of greater extent ; in some, tenderness of calf muscles. Three men, having marked pretibial œdema, and then sent to hospital, became later severely ill, and one died. Five others of the same force (of fifty) were admitted to hospital later in the year.

MacLeod has recorded a similar experience in Shanghai.

He says : 'Dr. Milles, of Shanghai, informs me that on the occasion of an epidemic in the Shanghai Municipal Gaol, he examined at the same time a number of Chinese hospital out-

patients, and was much surprised to find a striking proportion of them with numbness of the legs, with or without swelling, or patellar reflex, some of them merely complaining of not being well.'—*British Medical Journal*, August 14, 1897, vol. ii., p. 391.

On Christmas Island Durham¹ found that 'of 443 coolies not in hospital, 146 had diminished or absent knee-jerks, and 52 markedly increased knee-jerks. Moreover, just on 10 per cent. showed cedema of the shins, which was apparently independent of other causes than beri-beri.'

Many more cases, doubtless, occur in which symptoms really ascribable to beri-beri are never put down to their real cause. The patient has fatigue, or indigestion, which passes away, and of which he thinks no more. These larval or abortive forms make it all the more difficult to determine the real beginning of cases, which ultimately become acute, and it is a fallacy to believe the disease to have been incurred within any very short interval of exposure to whatever may be its cause, merely because it is for a short period only that the patient has recognised himself as being ill.

On the other hand (as in a case which is cited later), the cup which has been filling so slowly may at length suddenly overflow. Symptoms, insignificant to the patient at the onset, may be the prelude to an attack of appalling graveness and rapidity, through which the patient is reduced in a few days or hours to a condition of nearly total palsy, only too often fatal. Such 'pernicious' or 'fulminant' cases are difficult to distinguish from an acute myelitis, by whatever cause produced, or from the swifter cases of Landry's paralysis, which end in death.

Between these pernicious and the mildest manifestations of the disease every gradation exists, which according to fancy, or for convenience, may be classified as acute, subacute, etc.

The preservation in schemes of classification of the broad distinctions of type clinically observed is, in the writer's opinion, useful as indicating probably corresponding differences in the constitution of the original toxic cause.

H. Wright,² who, assuming an infectious origin for the disease, and adopting a short and quite arbitrary period of incubation, can see nothing more in it than an inoculation, which may or may not be followed by paralysis, has proposed to divide all cases

¹ H. E. Durham, 'Notes on Beri-beri in the Malay Peninsula and Christmas Island,' *Journal of Hygiene*, vol. iv., No. 1, January, 1904, p. 115.

² 'Studies from the Institute of Medical Research,' vol. ii., No. 1, 'The Etiology of Beri-beri,' p. 8.

8 THE CAUSE AND PREVENTION OF BERI-BERI

affected by beri-beri into merely two classes, one comprising the cases acutely or perniciously affected, 'marking the active stage of the disease,' which alone he would recognise as true beri-beri, and a 'residual beri-beric condition'—the paralysis.

His cases are thus grouped as :

'Acute pernicious beri-beri, which is always fatal.

'Acute beri-beri, which runs a course of from three to six weeks, leaving the patient paralysed.

'Beri-beric residual paralysis, which persists after the specific virus has ceased to act.'

The absence of any provision in this scheme for the slight attacks which Scheube has termed 'rudimentary,' and the occurrence of which is admitted by every authority, shows that Wright has not met with cases of the kind. Their recognition is nevertheless essential to a proper comprehension of the disease, the types of which, clinically, the writer would insist, afford the picture of a complete series of gradations of intoxication, from the slightest to the most fulminant effects.

Production of 'Chronic' Paralytics.—Whether the integrity of impaired nerves is ever completely restored, whether practical recovery is attained by the substitution for them of fresh neurones and fibres, or by the assumption, upon the part of sound muscles and nerve-fibres, of the work of those destroyed—a sort of vicarious hypertrophic compensation—the fact remains that most cases do recover. Those which do not do so, but which do not end fatally, leave the victim with muscles permanently useless,—not always because entirely cut off from the nerve-centres, or because wholly atrophied, but oftener, apparently,—because the sum total of the muscle-fibres left able to functionate is dynamically unequal to the strains they are required to overcome. These form the class of chronic paralytic beriberics.

In the majority of hospital cases the course of the disease is, after, as before, admission, gradual and progressive, whether towards death or recovery.

The event—the case-mortality—of beri-beri varies extraordinarily, both in different but perhaps neighbouring hospitals, which draw their cases from almost common areas, and in the same hospital at different times, without any difference in treatment by drugs or other factors¹ which could be held accountable for such mutations.

Although in the combination of the two main symptoms of

¹ The influence of *food* is not considered here.

the disorder—namely, dropsy and paralysis—every degree of gradation occurs, yet typical cases of the predominance of either exhibit, when regarded as syndromes of symptoms flowing from one and the same essential cause, a contrast so remarkable as to suggest that the difference in effect is attributable less to variability of reaction in the subject than to actual differences of constitution in the agent itself.

Such marked diversity of result is not known in the action of any living specific agent of disease. It seems incompatible with that of a single chemically definite body. It consists, however, with the behaviour of those natural toxic products which, although morphologically single, are compound or variable in composition, and correspondingly irregular in their effects.

Effect of Change of Environment.—No drug has, so far, been shown to be of any value in cutting short, or even relieving, the disease; but it is certain—this will be seen later to be a fact of some significance—that whereas cases remaining under the conditions under which they became attacked rarely recover without severe and prolonged illness, the progress of the disorder is often checked immediately, the rate of recovery accelerated, and the case-mortality of groups reduced, by the alteration of factors produced by mere removal to a fresh environment, or, as has appeared in numerous instances, by simple change of food.

The good effect of change of environment, which is admitted by all those having wide clinical experience of beri-beri (and the essential factor in which the present writer believes to be alteration of food), is denied by H. Wright, who says ('Study,' par. 498 [or p. 81]) he has 'never yet seen a case of acute-pernicious beri-beri recover after removal from the focus where the disease was contracted to a germ-free focus.'

Assumptions are made in this short passage—'focus,' 'germ-free focus'—for which there can be no possible ground of fact until the postulated germ which causes the 'focus' has become recognisable. But this apart, Wright's observation is explained very simply by reference to the definition of 'acute-pernicious' beri-beri given by himself earlier. It only includes cases which are *always fatal*.

Durham, discussing the same question, says¹: 'While I do not desire to combat the belief that a change of air, and *still more of food*, is beneficial, yet it seems that removal does not benefit the patient suffering from beri-beri to a greater extent than the patient suffering from many other diseases.'

¹ *Loc. cit.*, p. 138.

10 THE CAUSE AND PREVENTION OF BERI-BERI

The writer's view is that good effect nearly always follows change of locality, but merely because and in so far as a fresh (and non-toxic) diet is substituted for that formerly in use.

In this place the relation of such changes to alterations of locality is alluded to because a mass of evidence as to this sequence of events exists, the explanation of which lies in the food factor. (See Section IV.)

Symptoms always Symmetrical and Ascending.—The view generally held that the disease is wholly or mainly peripheral, or one at least affecting primarily the nerve-trunks or endings, scarcely harmonizes with the facts.

The prodromata of beri-beri are vague and *general*. The rapidly manifested peripheral effects are (with the exception occasionally of œdema) *always symmetrical* and (with still rarer exceptions, as regards commencing paresis) *always ascending*.

The location of the paræsthesiæ, the anæsthesia, the paresis, or paralysis, and subsequent atrophy of muscles, is *segmental*, not zonal—*i.e.*, the skin areas and muscle groups affected, in ascending order, correspond with the successive spinal roots or segmental levels of the cord attacked—not with the region of distribution of nerve-trunks.

The appearance of œdema first in the scrotum and perineal region, presided over by the lowest of the spinal segments, which is quite commonly observed, as also the occurrence of a paresis of bladder as the earliest symptom, are evidence of the ascending segmental effect.

Hypermyotonus.—The myotatic irritability, the superficial and deep reflexes, are first excited, and then depressed or abolished in like order. Contrary to what is usually asserted, the bladder is often early implicated, so that retention of urine occurs, but to the extent of paresis merely. It is never permanently or completely, or even long functionally, paralyzed. Sexual desire and capacity early disappear.

This is the picture of a spinal rather than a peripheral palsy, the result of a myelitis rather than a neuritis.

Durham¹ alone among students of beri-beri has laid stress on the hypertonus of muscles in the early stages. He says: 'Too little notice is taken of the preliminary exaggeration of the knee-jerk which so commonly precedes the loss thereof. . . . Observations on a number of patients showed that, given the increase, it may remain, and never disappear before the patient's apparent recovery, or it may slowly decrease to absolute loss,' etc.

¹ *Loc. cit.*, p. 133.

The writer has seen several cases in which clonus and rigidity of muscles in the lower limbs was present for a short time, and one at least in which typical spastic movement of the extremities persisted for more than a week before complete paresis occurred.

So far as discrimination according to type of the various forms of myelitis, poliomyelitis, or polyneuritis is of value, the position accorded to beri-beri nosologically, therefore, ought to be that of a myelitis with following nerve and muscle atrophy—a toxic amyotrophic spinal paralysis.

In the insidiousness of its beginnings and the final precipitancy of the disaster beri-beri resembles other general spinal maladies, notably, acute spinal paralysis of children or adults, Landry's paralysis, and (when they are acute) the conditions produced by alcohol, aconite, arsenic, and some other poisons. Most close is its likeness to Landry's paralysis. In both a rapidly developed, flaccid, symmetrical, ascending paralysis occurs, the result of a process involving gradually successive groups of spinal nuclei, but not the sensorium—in the case of Landry's disease, often the bulbar nuclei also. If in the latter disease the sensory disturbances are usually slight, that is often so also in beri-beri. If the bulb is more often implicated in Landry's palsy—the disease sometimes begins there—it is quite possible that this happens also, but is *overlooked*, in beri-beri. The recognition of deficiencies of speech, tongue-movements, of facial and ocular pareses, is difficult in patients afflicted with enormous anasarca, and whose distress precludes close clinical examination. But aphonia, difficulty of deglutition, loss of pharyngeal reflex, are often enough observed, and in one case at least Norman has recorded ptosis, strabismus, and dilated pupil.

The diseases are alike in the recoverability of the condition when arrested at an early stage.

Although a disease of the cord, it may be added there is, in Landry's paralysis, no impairment of the bladder. It differs conspicuously from beri-beri in the absence of electrical changes in, or of subsequent atrophy of, the affected muscles, and of oedema, though the latter is absent in a large class of cases of beri-beri also.

There are similarities, too, in the anatomical lesions, for, although the complete absence of all nerve-changes was made an essential point in the original description of his cases by Landry, they have been found by later observers both in the cord and in the nerves.

Acute Anterior Poliomyelitis allied Genetically.—In the acute

12 THE CAUSE AND PREVENTION OF BERI-BERI

anterior poliomyelitis of infants and others, the high initial fever, the sudden *completeness* of the paralysis, when discovered, and the asymmetry usually of its distribution, mark it off clearly as a syndrome distinct from beri-beri. But in the absence often of any prodromata, in the suddenness of its onset, its occurrence occasionally in epidemic form, and the essentially similar mechanism of its production as a partial myelitis, it presents features which must bring it into at least a genetic relationship with beri-beri.

Toxic Neuritis of Alcohol and Arsenic.—In the conditions which have been specially regarded as types of multiple neuritis—namely, the paralyzes due to alcohol and arsenic, and many other toxic and specific agencies too numerous to mention here—the clinical march of events resembles that of beri-beri so closely that Ross and Reynolds have considered it likely that the beri-beri of the tropics may be often really attributable to arsenic,¹ while Manson suggests that many of the forms of multiple neuritis frequently seen in temperate climates are really sporadic cases of beri-beri.² More will be said upon this head later, but here it may be observed that, without the pigmentation, and other skin lesions peculiar to arsenic (which are often absent), and without the general stigmata, and especially the psychic signs of alcohol, and in the absence of history of the application of either, some of the conditions caused by the two agents could not be distinguished from each other, nor either of them probably from the many forms of polyneuritis which, in the absence of preceding disorders, to which they could be ascribed as sequelæ, are regarded as idiopathic.

H. Wright³ has stated that the nerve-changes produced by beri-beri are indistinguishable from those due to alcohol, while it is probable that, were opportunity afforded of studying the lesions in cases dying of acute poisoning by arsenic, they too would correspond. Both these forms of so-called polyneuritis would then with beri-beri rank more properly as forms of toxic myelitis, with following degeneration of nerves and muscles.

The point likely to be of very great importance in beri-beri etiology emerges from consideration of these allied disorders, viz., that almost exactly the same syndrome of symptoms is the common result of many agents which act toxically upon the cord.

¹ *British Medical Journal*, October 5, 1901, vol. ii., p. 979.

² *Lancet*, 1898, vol. ii., p. 377.

³ *British Medical Journal*, June 19, 1901.

Asymmetrical Effects.—While symmetry of effect is the invariable rule in all the sensory and motor disturbances of beri-beri, the distribution of the œdema at initial stages sometimes contrasts with this. The following instance exemplifies this point, and affords a rare clinical proof that the incidence of the disease is primarily central, influencing soonest, in some cases at least, regions higher even than the cord :

A strong and otherwise healthy young native of India, who had been but three or four months in Malaya, came to consult me. He complained of having had slight 'fever' for four or five days, and being out of sorts, when he noticed swelling of the *left* side of his face, and as this was increasing, and he did not understand the cause, he came for advice. When I saw him first, the whole of the *left* side of the face was, as he said, considerably swollen. He had no bad teeth, neuralgia, deafness, facial paralysis, or any other local signs. I could make out no paresis, no anæsthesia about the face. There was that day no œdema about the legs, no anæsthesia there, or definite alteration of reflexes. They were sharp, and that of the triceps easily obtained. The muscles were not tender, but he felt 'weary' about the calves, and generally slack. I advised him that he probably had beri-beri, the more so as I learnt that he was feeding on uncured (Siamese) rice, a sort different from that to which he was, in his own country, accustomed. The next day the patient showed general increase of myotatic irritability, patellar, triceps, supinator, and wrist-extensor reflexes being exaggerated, and slight but distinct œdema about the *right* shin ; and the next three days witnessed an increase of this, until there was well-marked œdema, general over the whole of the *right side* of the body, leg, arm, and trunk. The left side of the trunk, left limbs, and right side of the face next became swollen also, so that the hemiplegic distribution became indistinguishable in general anasarca. Concomitantly, the usual train of other symptoms appeared, and this patient suffered from one of the very worst attacks not ending fatally which I have witnessed. He had complete paralysis of the upper extremities, which is uncommon, but none of the neck muscles, and fortunately no cardiac crises or diaphragmatic implication. All the deep reflexes had disappeared by the eighth day.

The crossed distribution of the œdema in this case shows that the first incidence of beri-beri intoxication is, in some cases at least, central. It is interesting also as showing that the vaso-motor or other machinery concerned in the production of œdema

is affected by apparatus situated above the pons, which is also unsymmetrical.

The complete symmetry in most cases of the nerve-lesions and symptoms, the presence (in œdematous cases) of the œdema in every tissue, are significant of the action of a poison universally diffused.

Where these effects are unsymmetrical they must be explained as the result of slight inherent differences in the tissues themselves, and in the case of nerve apparatus, at least, it is easy to conceive how one tract of cells should succumb earlier than another, if merely through more frequent function rendering it sooner exhausted.¹ The mechanics of the venous system may contribute to such an effect. The writer has at least noticed the occurrence in many cases of pretibial œdema upon one leg only, not always followed by implication of the other. In one such case, a European official of high position, such unsymmetrical œdema was noted upon several different occasions more than a year before a severe attack of beri-beri—the ascription of the condition to which cause at first had been scouted—ensued and proved fatal.

In the absence of definite local cause for it, such unilateral œdema must therefore be considered gravely significant, and as (in the tropics, at least) indicating always beri-beri.

It should be added that the sensory disturbances in the earliest stages, the anæsthesia especially, are not only segmental, but very often *at first* unsymmetrical. Some interesting diagrams which illustrate these points appear in a paper by Tiberio.²

Anatomical Changes.—While the parenchymatous degeneration of nerves in beri-beri has long been recognised, it is only recently that the central, probably always the first and essential, effects of the poison have been made clear.

Van Eecke in 1887³ described such changes as vacuolation of the motor cells of the anterior cornua.

Bentley, similar appearances in 1893.⁴

H. Wright described in 1901⁵ and 1902⁶ extensive changes affecting the cord, the peripheral nerves, the posterior spinal

¹ Compare with this the common incidence of lead-palsy on the *right* arm, and the case cited by Gowers from Bernhardt, in which, in a left-handed man, this condition was reversed (Gowers, 'Diseases of the Nervous System,' second edition, vol. ii., p. 948).

² *Ann. di Med. Nav.*, 1903, vol. i., fasc. vi., Giugno, p. 705.

³ *Gen. Tijds.*, 1887, vol. iv., p. 71.

⁴ *Beri-beri*, 1893.

⁵ *Loc. cit.*, *British Medical Journal*.

⁶ *Loc. cit.*, 'Study,' No 1.

ganglia, the stellate ganglia, and Meissner's and Auerbach's plexuses in the intestine. He states that in recent acute cases, hazy cells, with swollen and partially displaced nuclei or nucleoli, and slight chromatolysis, are to be seen in the ventral groups of almost every section of the cord, and in the lower part of the combined nuclei. In the dorsal horns he found such cells only in Clarke's columns. The posterior columns contained also a few degenerated fibres. The changes in the peripheral nerves were entirely parenchymatous at this stage, fibres being found in almost all nerves, but only near their terminations, in the early stage of Wallerian degeneration. 'There was no interstitial change whatever in either brain, cord, ganglia, or nerves.' In cases dying at later stages, which Wright terms residual beriberic paralysis, atrophic destruction of neurones was everywhere more marked, and there was advanced parenchymatous degeneration towards the ends of the trunks of most of the nerves, including the vagus. Except in the latter, there were also regenerating fibres.

The appearances, Wright says, are indistinguishable from those produced by alcohol.

Ellis,¹ in 1898, drew attention to a condition which had till then remained undescribed. Having paid particular attention to the appearance of the stomach, he found 'the lining membrane of that organ congested in thirty-one out of fifty-seven cases, in most of them intensely congested, especially on the ridges of the corrugations.'² In four cases there were blood-clots present in the stomach, no doubt from the persistent vomiting from which all had suffered prior to death.'

Travers states that von Holst,³ working at Kuala Lumpur in 1901, has also drawn attention to the same lesion as characterizing with particular frequency acute cases. Later, H. Wright has insisted on it as something almost specific. He, indeed, states his belief that the lesion is an essential feature of beri-beri. The disease is in his view nothing more than an acute infectious disorder, of which the stomach and duodenum are the seat of inoculation, the specific organism having gained entrance with the food.

Describing the autopsy of an 'acute-pernicious case' which had died after twenty days' illness, Wright says ('Study,' par. 462): 'The mucous membrane of the pyloric end of the stomach is markedly congested, and there are many scattered

¹ *Lancet*, 1898, vol. i., p. 1896, October 15.

² Durham wrongly attributes priority in this observation to Mott.

³ *Loc. cit.*

punctiform hæmorrhages on the edges of the valvulæ conniventes. [There were also petechiæ in the visceral layers of the pericardium and pleura.] A few duodenal glands are swollen and congested. The upper foot of the jejunum is in a similar but less marked state. There are no signs of the ankylostomum (*sic*) or its ova in any part of the large or small gut. [The patient was of a class—Javanese—who are generally free from this worm.] The large intestine is normal throughout. A large number of the mesenteric glands are moderately congested.'

'Par. 469 : I would especially draw attention to the condition in which the mucous membrane of the stomach and duodenum was found. The congestion was marked, and *indicates beyond all doubt the local action of a specific organism*. [The italics are the present writer's.] I have found this condition in all fatal acute-pernicious cases that have succumbed during the few weeks following the onset of the disease. I have always been able to exclude the ankylostomum duodenale. Taken in connection with the almost initial complaint of oppression in the epigastrium and loss of appetite, and the slight changes in the cells of Meissner's and Auerbach's plexuses, I feel bound to regard it as the primary seat of action of the specific organism of *beri-beri*, which has been ingested with the food [writer's italics], though not of it. It is significant that this lesion is absent in cases I propose to term beri-beric residual paralysis.'

It might be imagined from this that signs of local irritation and inflammation in the stomach could be produced by no poison save such as might be elaborated *in situ* by specific organisms.

The frequency of the lesion, and its proportionate relation often to severity of attack, may be accepted as a fact, the explanation of which, however, as it does not need, certainly does not justify the assumption that it is the initial lesion (or primary sore) through which the disease is acquired. The writer had as early as 1889 observed this appearance in many cadavers. But in nearly every subject ankylostomata were also found, to the effects of which, no doubt erroneously, as it seems now, most of the small petechial extravasations and erosions in the duodenum and jejunum were ascribed. The presence also almost invariably of masses of ill-digested rice in the stomach was held accountable for the signs of local irritation seen there. Patients suffering even from severe attacks, although at the onset of the disease they lose appetite, may feed greedily almost up to their last moments. Ravenous hunger, the consequence of the stomach condition, is often enough a symptom of the disease,

as it is of other dyspeptic conditions. The presence of food, swallowed often unmasticated, in a stomach already affected in its innervation, might well enough act mechanically as a local irritant; or, aberrant peptic processes ensuing, there might be formed some one or another of those ptomaines, the transition to which is easy from normal peptone. Inflammation and local necrosis might be due to these; or the lesion be purely dystrophic.

Abandoning these explanations, the stomach inflammation taken as a fact bearing a definite relation to symptoms of the disease, is evidence of an irritant at work which, in the light of the theory presently to be developed, it seems superfluous to regard as being other than a chemical poison, or as owing its origin to a source other than the food.

Viewed thus simply as a sign that material ingested by the patient then is, or recently has been, irritantly poisonous, Ellis's lesion—since he first particularly described it—is evidence supporting the theory that rice is the vehicle through which the poison causing beri-beri is usually conveyed.

Similarity of Ergotism to Beri-beri.—It is interesting, in connection with this matter, to recall the similar condition sometimes resulting from consuming ergot, the disease caused by which resembles beri-beri more closely than any other in its epidemiology, and to some extent in its clinical stages. Van Boeck describes, among other lesions due to spasmodic ergotism, 'strongly injected patches in stomach and intestine, sometimes hæmorrhagic and even gangrenous erosions' (Ziemmsen's 'Cyclopædia,' 1878, xvii., p. 891).

In animals poisoned by ergot Krysinski¹ found, in addition to necroses of intestinal epithelium, small hæmorrhages in the mesentery. In the cord there were changes in the postero-lateral columns.

The effect of poisoning by ergot, experimentally, is first to produce a rise, and then a fall of arterial pressure, with consequent venous engorgement and distension of the right side of the heart. In more toxic doses the blood-pressure steadily falls, as the result, apparently, of paralysis of the vaso-motor centre.

Mott and Halliburton² found the effects of blood taken from a case of acute beri-beri and injected into a cat to be first a fall of arterial pressure, with dilatation of the intestinal vessels and

¹ Quoted by T. M. Gibson, 'Text-book of Medicine.'

² *British Medical Journal*, 1899, vol. i., p. 265; *Transactions Royal Society*, 1899.

general venous congestion, the stomach, liver, and intestine being especially gorged, and the right side of the heart distended. In the liver were numerous microscopic hæmorrhages.

Ellis found that in 124 autopsies of beri-berics the heart weighed on an average 13.37 ounces, and that the right side was in every case much enlarged, while in 204 non-beri-beric cadavers examined concurrently with the others the weight was 9 ounces. This seems to be evidence that there is venous congestion in beri-beri which lasts a long time, sufficient, at any rate, to determine great compensatory hypertrophy. Ellis also found the spleen enlarged, being 9.27 ounces in the beri-berics of the same series, against 6.28 ounces for the others. This splenic enlargement is perhaps another result of the same venous stasis, and to the same mechanism it would seem reasonable to attribute, in part at least, the gastric and duodenal congestion, necrosis, and hæmorrhages, upon which so much stress has been laid by Wright.

Mott and Halliburton impute the toxic effects of beri-beric blood to the presence of bodies resembling, if not identical with, neurin and cholin. It is possible that, as Wright suggests, these are furnished by the breaking down of nerve-tissue which characterizes the disease, but proof of such a point must depend on determination which is the prior event—the nerve-decay or the appearance of a depressant poison in the blood.

In Landry's paralysis, Oppenheim states, in addition to some changes in cord and in nerves, hæmorrhagic foci in the bowels have been also described, with occasionally splenic enlargement.¹

In its pathology, as in its clinical course, therefore, beri-beri resembles other spinal disorders, and especially ergotism, with which Ehlers includes acrodynia.

Whether the resemblances of beri-beri to other myelo-neuropathies indicate real relationship of cause it is not necessary now to discuss. They are mentioned here as indicating the true position of the disease in classification, and to draw attention to the great likelihood there is that epidemics, and still more sporadic cases of any of them, may be mistaken for true beri-beri.

Other Diseases confounded with Beri-beri.—In practice difficulties have already arisen in temperate climates as to the discrimination of the polyneurites of arsenic or alcohol from beri-beri on the one hand, and 'epidemic idiopathic polyneuritis' on the other; and the mention of them must serve as a warning that not all cases, nor even all outbreaks of neuritis in epidemic form

¹ H. Oppenheim, M.D., 'Diseases of the Nervous System,' translated by E. E. Mayer, M.D., second American edition, 1904.

that are reported as beri-beri, even by able and unprejudiced observers, are necessarily to be accepted as such.

If the confusion of the disorders named above seems inevitable, it might be supposed that outside the class of *polyneurites* no other disorder could very well be confused with beri-beri, the broad outlines of which, as a clinical picture, are so distinct in typical cases—abrupt epidemics of paresis or paralysis, always with atrophy; never without some œdema; often with enormous anasarca; without any sort of ataxia or inco-ordination, tremors or rigidities, spasms or convulsions, other than cramps in skeletal muscles and the terrible angina of the dilated heart; with no mental complications and no definite or constant pyrexia.

Yet confusion has been made even by distinguished observers.

Ankylostomiasis, common scurvy, lathyrism, kala-azar, as well as true malaria cachexia, and anæmia of all kinds, and peripheral neuritis, such as commonly follows enterica and sometimes dysentery and other diseases, have all been mistaken for beri-beri.

'Epidemic Œdema'—constantly so Confounded.—A commoner, and in its results a more unfortunate error, has been the confusion perpetually made between beri-beri and the disease described by McLeod, Crombie, Lovell, Davidson, and others, as 'epidemic œdema.'

According to MacLeod¹ this disease, which prevailed severely at Calcutta and other parts of India in 1877-1878, and subsequently in Mauritius, and which Fayrer described as beri-beri, has this very obvious difference from the latter that there is never any paralysis, or even paresis. The principal symptoms are as follows: There is œdema, which is constant, and appears first, and sometimes only, on the front of the shins, and may extend to general anasarca. The urine may increase or be diminished, but there is no other urinary trouble, and never albuminuria; variable pyrexia initially; variable bowel complaints, of which diarrhœa is most common; causalgia—'burning in feet and limbs'—during onset, but no anæsthesia or paræsthesiæ; there may be skin-lividities and other eruptions occasionally. Nearly always dyspnœa, cough, palpitation, and thoracic, especially precordial, distress are present. There may be the worst cardiac angina. The patients are liable to onsets of this abruptly, and may die of it early and unexpectedly. Anæmia is

¹ *Transactions Epidemiological Society*, vol. xii., New Series, p. 55. Compare with this O'Brien's account of the same disease at Shillong (*Indian Medical Gazette*, 1879, p. 5), and Crombie's account of it at Dacca (*ibid.*, p. 114).

always present, and often severe. There is great emaciation, and prostration with it in the worst cases. It is slighter in others, but well marked in all. The duration is about two months. Anasarca often persists a long while. The case-mortality varies from 8 to 40 per cent. It attacks chiefly the poorer, but some of all classes. Paralysis is absent. Lovell¹ says it is never seen.

It is very probable that this complaint has an extended range.

Through the kindness of the Director-General of the Navy, I have been permitted to peruse records which show that epidemic œdema certainly prevailed for many years, and became finally severely epidemic among Kroomen at Ascension during the years 1895 to 1898, and that it was recognised by the men as endemic in the country of Sierra Leone, from which they had been recruited.

In Ascension the disease seems clearly to have been associated with, or favoured by, inferior diet, rice being most probably at fault, and to have ceased when more generous rations were provided than are usually given to natives. Most of the fatal cases in this epidemic showed great effusion into the pericardium, which often appeared at autopsy distended with fluid.

It is quite clear from the accounts of the observers—P. N. Randall and R. A. Ross, who treated most of the cases—that paralysis was not a symptom. Ross gives a graphic account of the terror and pain attending the sudden onset of the disease in its worst form—cardiac angina—in some of the victims.

In Mauritius the disease appeared in epidemic form in 1878-1879, and has perhaps been more or less prevalent there ever since.

In Diego Garcia, a small island dependency of Mauritius, whence all its stores and labour are obtained, Bolton² has reported the occurrence in 1902 of an epidemic which he describes as beri-beri, but all the stated features of which, as he gives them, correspond rather with epidemic œdema, or of that disease with a superadded scorbutic condition. In nearly all the cases there were signs of scurvy, and in all also œdema. The fatal cases mostly died with cardiac crisis, and had *œdema over the sternum*. Also there was swelling of the epigastrium sufficient to impress the lay observer who furnished the notes. But with the exception of one man, whose legs were swollen, who could hardly walk for pain, and another who, when inspected by Bolton, showed some weakness and wasting in a single leg, paresis, paralysis, and atrophy seem to have been absent.

¹ *Indian Medical Gazette*, 1881, p. 342; 1882, p. 148.

² *Journal of Tropical Medicine*, August 15, 1902, vol. v., p. 248.

Van Leent¹ has lately drawn attention to 'une forme mixte et peu connue de beri-beri et de scorbut,' cases of which he himself saw at Thursday Island. The health officer, Wassell, and the superintendent of the hospital at Port Kennedy, White, informed him that *all* the cases treated by them were of this mixed type. The men were mostly Australian natives engaged in pearl-fishing. The beri-beri showed itself both as 'hydropic' and as dry or atrophic. The diagnosis in these cases being that of Van Leent is hardly open to question, and there can be little doubt therefore that paresis or paralysis must have been among the symptoms.

The combination of beri-beri with scorbutus is, Van Leent says, 'd'ailleurs assez rare.' The writer has seen subacute scurvy in the Native States both in coolies with beri-beri and without it. In the former class a mild degree of gingivitis is fairly common.

Lastly, the fact must not be lost sight of that cases of Strachan's disease, the cardinal signs of which are an insidious paresis going on to general paralysis and atrophy, but without œdema, without paræsthesia, without cardiac crises, generally accompanied by some mental disturbance, never abrupt in onset or ending, and seldom if ever fatal, may occur elsewhere than in the West Indies, and lead to mistakes by the unwary.²

Salient Features of Beri-beri.—Among salient features of the disorder epidemiologically it may be noted that, though as a rule attacking chiefly males, it may affect—and in some epidemics has principally affected—females. Generally a disease of young adults, it may also attack children.³ It attacks earliest or more readily those affected with surgical lesions, and is apt especially to affect the lying-in.

In the Philippines and Malaya, among natives, Eurasians, and Chinese babas, it often follows after childbirth, and is then apt to be peculiarly fatal.

Clearly endemic in certain regions, upon occasions epidemic, it has never been anywhere pandemic.

Although seeming instances are recorded to the contrary, most epidemics have occurred among persons whose diet at the time

¹ *Arch. de Méd. Nav.*, vol. lxxix., p. 275, April, 1903.

² It seems probable to the writer that this malady may have occurred largely in South America, and in olden times have been a source of confusion with beri-beri there.

³ In Hong-Kong, Kuala Lumpur, and elsewhere, severe outbreaks have been recorded among female children.

was depraved, and whose systems had, through that and other causes also become so.

Though no occupation, no race, has any inherent immunity, epidemics of it in mixed communities show singular restrictions, attacking persons of one nationality, sometimes of one religion only, while sparing those of another living beside them and pursuing the same avocations.

Although certainly not contagious, the disease is easily transportable.

The 'infecting' agent, or the condition producing the disease, recurs so often in particular places—ships, dwellings—as to make it appear that they are permanently sources of its evolution, resisting measures of disinfection sufficient to thoroughly destroy germs.

No organism has ever been asserted, on any good authority to have been constantly found in the tissues of patients post-mortem. There is no uniformity among, and therefore no reliance to be placed upon, the many observations asserting the presence of bacteria in the blood during life.

That there is no definite period of 'incubation' or 'latency' seems certain. The time for which the cause may act before producing symptoms is probably very long as a rule, and is certainly irregular.

No meteorological factors form a constant in its evolution, or have been shown definitely to affect its epidemics.

It is certainly independent of soil, for it occurs afloat and on lighthouses remote from land.

No protection is afforded against a subsequent by a first attack. On the contrary, one predisposes to another.

Possessing features such as these, beri-beri falls naturally into the group of diseases which are produced by general poisons or intoxications. Such agents, absorbed in single or a few large doses, may almost immediately produce overwhelming and destructive effects, or may, through the accumulative action of small quantities continually administered for long periods of time, entail the same results.

Although generally diffused, as they must be by the circulation throughout the system, from the organ by which they are absorbed, such poisons exhibit specific effects in the tissues which they attack, owing rather perhaps to intrinsic qualities or defects of the latter than to differences in the distribution of the agent itself.

The nearest congeners of beri-beri thus viewed are therefore Landry's paralysis, the so-called multiple neuritis ascribed to

alcohol and to arsenic, the paraplegic forms of plumbism, ergot-poisoning, lathyrism, and pellagra. With the intimate anatomical lesions described as characterizing the former members of this group, those found in beri-beri present close resemblance, and both in this respect and in its epidemiological features the parallel between it and especially ergotism and pellagra is striking.

Primâ Facie Incompatibility of its most Marked Features.—

To epitomize, the most notable feature about the disease is the *opposition* which its most marked characters bear to each other.

1. It has seemed, and is still held by many, to be essentially a local disorder, a 'place infection,' in the sense that occupants of particular, and even confined localities (small districts, houses, ships) only are affected by it, while those living immediately around them, under precisely similar outward circumstances, remain healthy. Yet the cause is certainly transportable, and new 'foci' of the complaint appear to have been set up in more than one instance by immigrants into places where it had formerly been unknown.

2. It is, there is ample evidence to show, not infectious, assuredly never directly contagious; yet it has, in rare instances, unaccountably followed the introduction of persons affected with it, and spread among communities previously free.

3. Its transportability and extension in new places argue a living multiplying cause, but its clinical course negatives the view that any such agent multiplies in the system, since the vast majority of cases exhibiting early symptoms do not progress, but entirely recover, especially upon changing the circumstances under which the disease was acquired.

4. Finally, although neither race nor age nor sex confers immunity, although it is known to be uninfluenced in its incidence and course by occupation or by climate, and to be independent of the dietetic value of articles consumed, yet in epidemics in communities of mixed nationality it constantly happens that individuals of a special religious caste or status or particular races only are attacked, others wholly escaping.

The theory, therefore, which is to explain beri-beri must reconcile many facts apparently contradictory.

This unconformability, even contradictoriness, in its characters, inexplicable on the hypothesis of its cause being an agent which should be specific, and whose action, after its incidence upon the individual, should produce definite and constant results, argues the *variability* of one, if not more, inconstant factors in its causation. The characteristics of beri-beri are, in fact, such as would

belong to a disease dependent upon the concurrence of *two* agents, or two groups of conditions, for its causation—those, namely, on the one hand, specific of, or peculiar to, the active causal agent itself, constant within the limits which the physical conditions of its environment impose upon it—the ‘specific’ extrinsic factor; and, on the other, something which place and season, occupation and nationality, might or might not modify, something which should be capable, if not of directly modifying the effects, yet of determining the chance and extent of incidence of the effects which the extrinsic agent could produce, something as irregular in its manifestations as would be the exercise of *volition*, the dispositions of which, all-important as they must be in modifying the incidence of the extrinsic factor upon the patient, nothing in telluric or meteorological conditions can very well control—in short, a personal condition, a habit, or custom of the race or individual.

Now, there are only two extensive factors of disease to which man is liable, against which he can naturally effectively guard himself, or the incidence of which he does actually modify by the exercise of the personal factor, the operation, intentional or unwitting, of habit and custom—viz., (1) the attacks of hostile living organisms, and (2) impurities in food.

Nothing of the nature of evidence is at hand to make the former class of agents probable as a factor in beri-beri, but to prove this it will be necessary to consider various theories which have been propounded one by one.

1. Theory of Bacteria in the Blood—Experimental Tests.—

To the conception of the disease as due to the presence of *bacteria in the blood*, which is still admitted by Manson as having some vogue and vitality, may be opposed the following observations of the writer, as reported to the conference of medical officers of the Federated Malay States, held at Kuala Lumpur, November, 1900:

‘Three hundred and forty-seven cultures were made in all from the blood, skin, nerves, and other tissues, living or dead, of beriberics, and healthy persons used as controls. . . .’ Of these, eighty-one experiments, recently made, were particularly dealt with, as showing that, ‘when a sufficiently rigid technique was observed, no bacteria of any kind are to be obtained from the blood of beri-beri patients.’ The better to illustrate the fallacies underlying this mode of research, the experiments were divided into two groups.

In the first, separate cultures were made from the skin

and living blood of each of thirty-three people, of whom twenty-five were typical beri-berics, taken at as many different stages of the disease as possible, and eight were controls, free of all symptoms. The skin was first thoroughly scrubbed with spirit soap, next with a strong sublimate solution (in ether and alcohol), and finally wiped with a sterilized plug soaked in alcohol. Blood was then withdrawn from the vein with a sterilized syringe, which was washed out with sterilized water and boiled again before use on the next case. Several syringes were used. With blood from the syringe, after expelling a first portion, broth tubes were inoculated and incubated for twenty-four hours at 99° F. Subcultures were made from these on agar and gelatine.

The result was that, of twenty-five beri-berics, growths appeared in the blood-cultures of twenty-four. But of eight controls, there were also growths in six.

The growths obtained with the blood were similar in both series. As often as not more than one species appeared. They belonged to the staphylococcus group, resembling *Micrococcus pyogenes albus* on agar, but liquefying gelatine much more slowly; and agreed with the described flora of the deeper skin. The principal forms obtained resembled closely, if they were not identical with, *Staphylococcus citreus* and *flavus*, *M. tetragenus*, and *S. epidermidis albus*.

In the next group of cases the procedure was designed to exclude entirely contamination by these air and skin flora.

A separate, new, and thoroughly sterilized fine injection-needle was used for each case. The skin was cleansed as before, the needle plunged into the distended vein, unattached to any syringe, and the blood allowed to flow through some moments before the media were inoculated. A few drops were then let fall direct from the vein into each tube.

Tested in this manner, the blood of twenty-three persons proved absolutely sterile (the tubes were shown to the meeting). Sixteen of these were typical beri-berics, and seven controls.

These cultures were aerobic. Repetition of the experiment upon another series of twenty-five cases taken at different stages of the malady, making anaerobic subcultures, had the same result. All the bloods examined proved absolutely sterile.

The writer thus became convinced—and anyone who will repeat the experiments in the same way may attain the same conviction—that there is not to be found any coccus or bacterium in the blood of beri-berics at any stage of the disorder which will grow upon ordinary media.

Stanley in Shanghai, and Durham, later, in Selangor obtained similar results.

Beri-beri is not a 'microbiohæmia.'

Dangerfield, the latest writer on beri-beri, must be added to the long list of those who, having obtained growths with blood derived (at uncertain stages of the disease, because the specimens were sometimes sterile) by pricks through the skin of beri-berics, believes beri-beri to be due to such an organism, and *therefore*, he at once assumes, also infectious and contagious. Dangerfield, whose observations were made in Réunion, made many inoculations upon various sorts of animals with the micrococcus he derived from finger-pricks, and also from the tissues of the dead body, with excreta and sputa of beri-beri patients. Some of these animals died with signs of paralysis. Two veterinary surgeons certified such a paralysis in animals submitted to them to be true beri-beri (p. 208). But it is remarkable that Dangerfield shows no sections of any human tissue with his micrococci in them; and, what is more significant, that the so-called pathogenic cocci which he sent for examination to the Pasteur Institute proved, in spite of every precaution taken in sending them, to be, like Lacerda's organisms long before, non-pathogenic in Paris. This, he would have us believe, is to be attributed to the effect of the colder climate on the organism! Dangerfield, I must add, appears to make no distinction between *epidemic œdema* and beri-beri. Indeed, the former malady, although it almost certainly occurs in Réunion, is not even mentioned in his work.¹

2. Wright's Theory of Dirt Infection.—Among recent investigators, H. Wright has formulated a sort of botulistic modification of the infection theory, whereby patients are supposed to carry about and deposit, and continually infect themselves and others with a germ which they pick up in dirt, and convey into their food, and pass again in fæces. The germ is non-volatile, and has not been revealed by bacteriology.

This is, of course, nothing less than to postulate a *contagion* by which, while there are common natural laws in operation, the dirtiest classes must become most infected, and as a result of which those who bring beri-beri into countries previously free from it should leave the disease everywhere in their train. But to these conceptions the whole epidemiology of the disease is opposed. In Malaya it is the dirtier classes—the Tamils, who in

¹ Dangerfield, H. Vivian, 'Le Beri-beri,' etc., Paris, 1905, A. Maloine.

particular are extremely foul in their habits (they defecate promiscuously anywhere within a few yards of their dwellings)—who almost alone and uniformly escape beri-beri. The Chinese, comparatively careful in this matter, furnish 96 per cent. of the victims, and among them the sinkhehs, who bathe daily, are most attacked.

Wright bases his views of the infectious nature of beri-beri upon certain experimental results obtained by himself at Pudoeh Gaol in Selangor.

Having selected all the healthy prisoners passed into that institution during a term of eight months, he divided them into two unequal parties, located these in different parts of the prison, isolated them from contact with each other or other possibly 'infected' persons, and fed them all on the same 'physiologically correct' diet. Fish of every kind was excluded, and the rice given was effectually sterilized daily. 6.4 per cent. of the larger party (90 cases among 1,406 individuals) acquired beri-beri under these circumstances. None of the smaller party were affected.

On this showing he concluded against the possibility of beri-beri being caused through any kind of food, particularly rice. Since an air-borne infection or general emanation would not, in his view, harmonize with the escape of the small party, he assumes the only alternative—that of a contagion intimately connected with the immediate surroundings of each patient, and requiring his own agency for infection; in short, a filth disease, the result of dirt ingested accidentally with food.

Into this position Wright seems to have been driven by the force of two gratuitous assumptions: (1) That all who share the same diet must, if it be poisonous, become equally affected by it; (2) that the sterilization of food (rice) must deprive it of all poisonous qualities.

Fallacies so egregious condemn any conclusions based upon them.

The common example of alcohol illustrates familiarly the extraordinary difference there is in the susceptibility of persons to identically the same poison. Allbutt says of *ergotism*: 'Often in epidemics some members of a stricken family escape; other patients are sporadically victims in the midst of a general immunity.'

Were such a poison as ergot present in rice it would certainly not be destroyed by boiling, while in the case of many alkaloids it is even likely that superheating under pressure would greatly

aid their solubility. So long as the (postulated) poison in rice which causes beri-beri is unknown, it is more than idle to state that boiling rice deprives it of its toxic properties. Moreover, the numerical probabilities were greatly against Wright's smaller party getting beri-beri during his experiment, since it contained a daily average strength of only 34 men against 221 in that in which the disease appeared. In the latter only 6.4 per cent. of the individuals were attacked. It was therefore 15 to 1 against any individual admitted to the gaol acquiring the disease, and 6 to 1 against such a case being included in the smaller party.

No evidence locating the presumed infection in particular cells is offered by Wright, yet, as only 1 in 15 of his affected party got beri-beri, some evidence in this direction ought to have been procurable were there any truth in the hypothesis.

He considers that his view is confirmed by the appearance of symptoms resembling beri-beri, and a demonstrated polyneuritis in certain monkeys which were confined in the cells, but not fed on rice. In the case of which he gives detailed account septic infection is admitted, and Durham shows reason to believe that this was the case with all of them.

Wright states: 'The food of all these monkeys was bananas, sugar-cane, and pine-apples. Before being fed (*sic*) to them the fruit was *rubbed on the floors to pick up the specific organism I assume is present*' ('Study,' p. 63, par. 387). It clearly would have destroyed this object had their cells been cleaned. Durham¹ describes the result as 'nauseatingly filthy.' It is not surprising that, under such conditions, illness supervened.

Oppenheim has described such undoubtedly septic cases of polyneuritis, and goes so far as to hint that many forms of peripheral neuritis following specific complaints may own a similar origin. Durham,² moreover, states that monkeys of the same batch as Wright's, not confined in cells, and 'not exposed to any presumable beri-beric influence,' grew similarly diseased, and at autopsy showed extensive nerve degeneration. One of these, like Wright's, became infected from a surface abrasion, and had pyæmia—liver abscesses. In another abundance of staphylococci were found. 'Both these, as others in the same batch, were found to be infested with a malaria-like parasite in their red blood-corpuscles.'

It is worth while adding that according to Wright's own

¹ *Loc. cit.*

² *Loc. cit.*, p. 128.

account the polyneuritis in his monkey appeared to differ from what he found in a case of human beri-beri after illness of exactly the same duration. In the man only the *ends of the nerves* near the muscles were affected, and slight early changes throughout the cord were general. He makes it a point that 'in acute-pernicious cases one never finds atrophy in the main trunk of any nerve.' In the monkey almost every nerve-trunk is described as being affected almost throughout its whole length.¹

3. **Durham's Theory.**—Durham is the latest among the supporters of an infectious origin of beri-beri, on the ground chiefly, as it would seem, of the difficulty of reconciling the apparently conflicting facts known about the relation to it of food and rice.

He offers no new evidence of any kind in support of his view, but cites several instances with difficulty intelligible on an infection hypothesis, and has observed a faucial condition which has suggested to him the possible analogy of the disease with diphtheria and its sequent neuritis.² 'This infection,' he says,³ 'is not of the nature of a septicæmia (since the internal organs at death prove sterile), but to (*sic*) a surface condition about the upper air-passages. From the observation of the throats of a number of patients it is surmised that the redness which is seen therein, especially in early cases, may be intimately connected with the disease.'

One would have supposed that the claim on this ground should have been for a septicæmia due to absorption of products from a local surface, as opposed to a pyæmia or general dissemination of the infectious body.

Durham grew peculiar 'looped' colonies of streptococcal forms from such throats, and in a single one out of several monkeys 'infected' by throat swabbing from a beri-beric got a semblance of beri-beric symptoms. But in view of his demonstration that monkeys easily succumb to peripheral neuritis of septic or other

¹ Wright has been kind enough to ascribe to me ('Study,' p. 60, pars. 371-377), and has found it easy to demolish, the theory that beri-beri is 'due to the ingestion of a specific organism which develops on growing rice.' I trust it may not appear ungrateful if I hasten to disavow paternity for the view stated in this form. My belief is that beri-beri 'is due to a *poison* found in rice, which is the result or the specific product of some organism—epiphyte or parasite;' but I do not believe the disease to be due to the ingestion of the *organism*. Nothing which I have said, written, or published affords ground for the misrepresentation.

² Bentley had suggested this earlier.

³ *Loc. cit.*, p. 153.

origin, little importance can be given to the slender presumption afforded by this one experiment. The specific objections which this observer lays against *rice* as a possible cause of beri-beri are easily disposed of, and will be dealt with later.

4. **Theory of Co-operating Factors.**—Most of the observers who support infection believe everything in beri-beri epidemiology to be explicable by such means. By them the evidence of such facts as may have been established as to the dependency of the disease in some cases upon food as at least its medium is not investigated; it is simply denied.

Vorderman, Legrand and Burot believe it may be possible to reconcile both these two views—dependence on food and causation through infection—the influence of each factor being accepted by them as proved by competent witnesses.

Their implication seems to be that, just as some other admitted infections—typhus (famine fever) or relapsing fever—occur chiefly under certain conditions unfavourably affecting the individual, so beri-beri is caused by an infection which, however, only operates effectively when the system upon which it is inoculated is in a special state, such as is induced by eating certain unwholesome sorts of diet.

This, as a vague possibility, is, of course, a theory difficult to dismiss; but it may be sufficient to say here that it will be shown first that the evidence is against any sort of infection causing beri-beri: it is certain that so far no organism of any kind has been isolated in the system, or found to occur more commonly in the condition which causes the disease; secondly, that there is ample evidence of the production of beri-beri through food alone.

Travers' Experiment.—Against the possibility of the disease being due to *any form of infection*, an experiment conducted by Travers at the two gaols in Selangor offers evidence difficult to explain away.¹

Beri-beri having broken out among the prisoners at a newly-occupied gaol in August, 1895, it was decided, for the purpose of testing, not the question of infection, but that of food, in relation to it, to keep a proportion of the prisoners at each of two gaols—the old and the new. Accordingly, between October 21, 1895, and June 30, 1896, a period of eight months and nine days, two-fifths of the average daily strength of the prisoners were kept at the old and three-fifths at the new prison. Throughout that period they had identical diet, and all other circumstances of

¹ See *Journal of Tropical Medicine*, August 1, 1902.

work, exercise, etc., were alike. During the term there were some 2,500 committals to prison, and among the prisoners 86 had beri-beri—many of them in acute stages—*on entering the gaol*, 193 developed it for the first time within the gaol, and there were 107 'relapses.'

There was, therefore, a constant source of (assumed) infection in the persons passing into the prisons, as well as possibly within the prisons themselves.

Now, if beri-beri be due to any form of infection multiplying in the body, it must be granted that, once effectually lodged within the system, it can in no way affect the progress of the disease to maturity, whether the subject enter prison or not, nor whether he be lodged in this or that gaol.

Again, whatever the period of incubation assumed for the disease, it is certain that there is a stage at which, although the inoculation (or infection) has taken place, the symptoms following from it have not yet become developed, are not evident—for instance, at the actual time of admission to gaol.

Since it is a fact that numerous persons did pass into prison at all stages of the disorder, it follows that, if not an equal proportion, some at least among those admitted were, though apparently healthy on admission, in reality then already in incubation stages of the malady.

In such persons it is obvious that the symptoms must have appeared after admission, to whichever of the two gaols the subjects should have been assigned. However the prisoners were divided between the two gaols (provided that the number at each was sufficient to form a reasonable basis for conclusions at all), it must have happened that some of such 'larval' cases should have matured, have fully developed the disease at each place—at the old gaol, therefore, as well as the new.

But this did not happen.

Although two-fifths of all the prisoners, forming a daily average strength of more than one hundred persons, were kept under observation at the old gaol for the eight months of the experiment, although the apportionment to each place was made without any intentional selection, except to exclude those already affected with the disease, although altogether 386 attacks of beri-beri were noted among the prisoners as a whole during that time, yet as a fact, according to Travers, *not a single new case of the disease originated at the old gaol.*

Does not such a result offer convincing evidence against the

possibility of this disease being caused through any form of infection?

5. **The Pure Miasma Theory.**—There remains what may be called the pure miasma theory. The extrinsic cause, it is conceived—a mould, a microbe—produces a toxin—dust, liquid, a gas—the continual absorption of which, disseminated or spread through air into the system produces beri-beri. This conception is especially favoured by Manson.¹ He suggests the parallel of alcohol produced by yeasts. Similar organisms, in places where beri-beri is endemic, would explain particularly well the supposed continuous infectivity of such places as houses and ships.

An even exacter analogue for such a means of origin would be arsenic, as it is absorbed from poison-papered walls. Organic gaseous derivatives of the metal, far more toxic than itself, are liberated from matters containing it by the action of moulds (*Penicillium brevicaulis*, according to Gosio, Saccardo, and others) which are thus to be regarded as the prime agents in the causation of the disease. By just such an agency as this, it is claimed, can the apparent and so often, it would seem, very tenacious 'place-infectiousness' of beri-beri be explained. The germ lodged in the floors or upon the walls of some building or ship distils slowly a poison, to the effects of which those who inhale it day by day sooner or later succumb; or, after long remaining inert, under the stimulus of some change or other in the physical conditions there is manifested the same result.

Alluring as this conception may be, applied to explain the limited class of instances of what may for the moment be termed 'house' or 'room' beri-beri, which it appears to fit, it is easy to see that it is wholly inadequate for the great majority of cases, which are acquired under other and very different conditions. Among Malays, for instance, beri-beri never occurs while they live under the primitive conditions customary in their own homes. It is usually incurred when they are abroad upon jungle expeditions, not occupying any house, nor, indeed, the same camp from day to day. Is it credible that every spot occupied during their itinerary should be the seat of the same sort of germs, distilling constantly the same deadly toxin? Were this supposable, the next postulate to be granted must be that the toxin, gas, or whatever it may be, is generated in such quantities at its source as to be still capable of poisoning even after free dilution by all the winds of heaven.

¹ *Lancet*, November 23, 1901, vol. ii., p. 1391.

The tens of thousands of cases of beri-beri among Chinese mining coolies with which we in the Malay States have annually to deal, again, come from buildings which are, as a rule, freely ventilated, and which, moreover, so far from having become soaked or contaminated with old infection, are generally recently put together, and of new, even sap-green, materials. Indeed, the most numerous and the worst cases usually occur in those mines which are farthest afield and most lately opened, and in which the dwellings are, in consequence, quite new. In this case, no doubt, the germs would be held to be lodged in the ground. Beri-beri prevails, too, in absolutely fresh brick-and-mortar, properly ventilated, dwellings, from the first date of occupation.

Most remarkable of all, it appears in mid-ocean upon ships which have never been in countries where there was beri-beri, and whose crews have never been in contact with others who may have had it.

The disease is not, like influenza, carried—it is not conceivable that it should be carried over immense distances of ocean—by air. It is not contagious; it is conceded (by this theory) to be not even infectious. It cannot, any more than any other disease, arise *de novo*: it must, therefore, have been introduced into these ships artificially. To fit the 'miasma' hypothesis, it has to be conceived that the pathogenic agent is thus brought by persons who have never suffered from it and whom it does not at the time affect, from places in which there is no other evidence of its existence, to be implanted in a ship, in which, again, it grows with such capricious, but at the same time deadly, effect that upon one occasion perhaps all the fo'c'sle hands shall be attacked, and may die, upon another the officers of the cabin mess alone, and on yet another occasion both officers and crew shall succumb.

But, in spite of quoted and notorious seeming instances to the contrary, it is not *common* for beri-beri thus to 'cling' to quarters where sufferers from it have been housed. Nor, certainly, is it a disease spread by personal contagion. Hundreds of hospitals, the attendants of which remain free although in close contact with beri-berics daily, and in which no 'focus' of infection is ever set up, attest the contrary.

The appearance of 'predilection for particular ships,' which some observers have persuaded themselves beri-beri has, is probably a fallacy based on insufficient figures. The instances in which outbreaks recur on the same ship are recorded because they greatly impress the observer, but they bear only an insigni-

34 THE CAUSE AND PREVENTION OF BERI-BERI

ficant proportion to the total number of vessels upon which single outbreaks happen which are never repeated.

Rees,¹ who in particular advocates the connection of the poison of beri-beri with ships themselves, cites statistics which seem to the writer to lend very little support to that conclusion. Dealing with the vessels of a particular line trading to the East, he states that between August, 1890, and August, 1897, 157 cases of beri-beri were admitted to hospital from *one-half of the total number of ships engaged*.

13 ships	had 1 outbreak	each
13 "	" 2 outbreaks	"
7 "	" 3 "	"
5 "	" 4 "	"
2 "	" 5 "	"
1 ship	" 6 "	"

Total 41 ships attacked out of a fleet
of twice that number.

Rees suggests that 'these figures afford evidence that beri-beri has a predilection for particular ships.'

But if a portable agent of disease be postulated, whatever its nature, and whatever the means by which it is brought upon the ship to cause an outbreak, it is surely easier to assume that the same extrinsic train of events which determined the outbreak on the first is *repeated* on the second occasion than it is to believe that the second outbreak is the result of the pathogenic agent having become attached to the ship?

The simpler view is, moreover, justified by the figures given.

Of the whole fleet of eighty-two vessels, the disease occurred in but one-half—forty-one. The chances that any particular ship should or should not get the disease were originally, therefore, equal. The chances of a ship once attacked becoming so again are not, however, diminished by the first attack, and for each ship, therefore, the chance of getting it on subsequent occasions remained as before—equal. On the basis of such mere chance alone it may be calculated the number of recurrences on each ship would have been much the same as was actually observed. The numbers of ships which showed recurrences, out of the forty-one originally affected, *according to observation*, were :

Second, third, fourth, fifth, sixth epidemics.

27 15 8 3 1

¹ *British Medical Journal*, 1897, vol. i., p. 747.

According to calculation, they would have been :

Second, third, fourth, fifth, sixth epidemics.

20 10 5 3 1

It is further to be observed that the calculation shows merely *the chance of the disease being imported afresh* on each occasion ; but for ships already once infected—if there be anything in the place theory—the chances of recurrence should be something much greater.

An infection which has been shown capable of persisting on a single vessel throughout six voyages ought, other circumstances being equal, to do so in all. Recurrence should be the rule. But, as a matter of fact, the figures show it was rather the exception, for it recurred *oftener than once* in one-third only of all the ships into which it was introduced, in a third *only once*, while in yet a third it *never recurred* at all.

In these remarks I have assumed, since Rees did so, that all the vessels dealt with were, as statistical units, equal. This was, of course, not really so ; such a state of affairs would be impossible in practice.

The length and season of the voyage, the nationality, and even the religion of the crew (upon which their food-habits often depend), the nature of the provisions used, and their relative quantities in the rations issued—all these are factors which, it will subsequently be seen, actually do modify the incidence of beri-beri upon communities, at sea as upon land. These conditions, therefore, must be shown to be the same in any ships under comparison before conclusions drawn from the prevalence of disease among them can have the slightest value. With the ship must be considered all that it connotes.

Miasma Theory Incompatible with the Inequality in the Incidence of the Disease.—One fact above all seems to make the miasma theory untenable. It does not explain—is, in fact, clearly incompatible with—*the exceeding inequality of incidence of the disease on different sections or classes in communities of persons all equally susceptible and simultaneously exposed to its influence.*

Sometimes the dividing line follows race, sometimes social position or status, sometimes religion, sometimes even a mere personal difference, such as marriage.

In Brazil Féris¹ says it has been observed that ' the persons

¹ Féris, *Arch. de Méd. Nav.*, June and July, 1881, August, 1882, t. 37, 38, pp. 4, 66.

36 THE CAUSE AND PREVENTION OF BERI-BERI

most attacked are those of the higher, rather than of the lower, classes,' a fact for which there is offered no explanation.

Simmons,¹ Baelz,² and Saneyoshi³ have noted a similar incidence in Japan.

Brémaud⁴ relates that in an epidemic which he witnessed on a ship—the *Ilione*—conveying Indian coolies from Pondicherry to Martinique, the Mohammedans only were attacked, who did not eat pork, whereas the others did, a fact to which he attributes their exemption.

In the Rangoon Hospital Barry⁵ states that of 871 Indian coolies admitted to hospital for beri-beri between 1896 and 1899, 769 were Hindus and 102 Mohammedans. That is to say, the disease was confined among Tamils to those of strict caste, although in any 1,000 Tamil coolies of the class represented by immigrants to Burmah quite a large proportion would be classified as of no caste, or of no strict religion.

In the great epidemic at and around Manila in 1881-1882, according to Königer,⁶ Tagals (Malays) were almost exclusively attacked, Chinese very rarely, Europeans and Eurasians not at all, although the latter classes were numerous, and all intermingled. Europeans were similarly exempt in the epidemics there recorded by Smart⁷ in 1902-1903.

In an account of an epidemic related by Lasnet⁸ among convicts in the Dakar Prison in Senegal, where Europeans and natives were confined together, although forty-five cases occurred among the latter, not a single European or Mulatto was attacked.

The same relative though not complete immunity of Europeans as compared with natives has been noted by Rupert in Borneo, Van Leent and others in the Dutch Indies.

In the epidemic described by Rowell at the Singapore Gaol from 1875 to 1886, only the natives, not the Europeans, were attacked, and the same fact is noted in the epidemic prevailing in the same gaol to-day. The wards were, and are, however, contiguous, and all other hygienic conditions are alike among the Europeans and natives. In that and similar outbreaks, as in

¹ *Med. Rep. Imp. Mar. Cust.*, 1880.

² *Zeitschr. f. Klin. Med.*, 1882, vol. iv., p. 616.

³ *Sei-i-Kwai*, May 30, 1901.

⁴ *Arch. de Méd. Nav.*, 1899, No. 5, p. 369.

⁵ *Indian Medical Gazette*. September, 1899; May, 1901.

⁶ *Deut. Arch. f. Klin. Med.*, 1884, vol. xxxiv.

⁷ *Report Surgeon-General Army U.S.A.*, 1902-1903.

⁸ *Arch. de Méd. Nav.*, 1897, pp. 138, 210.

the Selangor (Pudoh) Gaol, at the present moment, the convicts alone are attacked ; the warders, also natives, but not of the same race, escape. In the Australian pearling fleets, it was noted by Haynes,¹ the 'South Sea men' and the Australian natives were 'entirely proof' against beri-beri, as were also the Europeans. The Malays and Japanese succumbed. In the matter of housing and exposure the conditions were, of course, the same for all the crew.

Finally, as I shall presently show, in the Straits Settlements and on the adjacent Peninsula, throughout the whole area of which the disease is endemic, among a mixture of immigrants of different races, living under precisely the same external conditions, it uniformly spares two—the Indians and Europeans.

The relation of Malays in the Peninsula to the disease furnishes proofs against the possibility of its being due to 'place effect,' which are very numerous. For the opening up of the country has entailed the occupation of innumerable spots all over its area by parties of Chinese ; they are engaged both in agriculture and in mining, while there is not a village in which some of them do not squat as hawkers and petty traders. Sometimes the mine or estate is opened up in virgin soil, previously uninhabited jungle ; sometimes the intrusion is into areas already occupied and cultivated by Malays. It matters not to the result whether the newcomers have beri-berics among them or not on arrival, or whether they are all (as is usually the case in opening up new mines and estates) healthy men, mostly coolies newly immigrant from China. It equally little affects the issue that the disease may have been wholly unknown in the locality previously. Sooner or later, generally within a few months, beri-beri appears among these Chinese, whatever their locality and whatever their occupation.

The experience of the writer, who has closely studied the conditions of all classes of operatives and settlers in the Native Malay States since 1889, leads him to assert that *this event is invariable*. It would be impossible to discover an instance of any considerable body of Chinese coolies (a score or more) engaged at work in any part of the country for more than six months among whom some cases of beri-beri had not occurred.

It is a commonplace of local observation that beri-beri thus breaks out—'lights up,' as it is said, in 'new foci'—among Chinese, and even imported Malay (Javanese, Boyanese, Bangoereese) occupants of districts, villages, even houses, the previous

¹ *Journal of Tropical Medicine*, March, 1900.

inhabitants of which, whether Malays or others, continuously resident in them for years, and perhaps generations, under exactly similar external conditions, had as constantly escaped the disorder, and, what is more, continue to remain so exempt even while it actively decimates the new-comers.

The susceptibility of the Malay to beri-beri, even if not, as there seems evidence to show, greater than that of the Chinese, is certainly not less. It is improbable that in all these instances, which time and space have multiplied to thousands, the Malays should escape by mere chance. It is impossible that if the cause of the disease were active in the place in which they were dwelling, it should uniformly fail to attack them. Their primitive freedom from it, and their continued exemption even while living in close contact with their affected neighbours for months and years together, prove not only that the cause of the disease is imported, but also that it is one the influence of which, when it is imported, they do not share.

It would be manifestly impossible for the Malays thus to escape effects which were indigenous and due to place alone.

Now, all the different races noted as escaping beri-beri in the instances quoted are known to be readily susceptible of it under other circumstances. A scourge of the Dutch (Malay and European) army and navy in Sumatra, affecting Polynesians in New Caledonia, Australians in New Brunswick and Australia, a grave cause of mortality among the Indian immigrants into Burmah during the rice-harvest—it is clear that exemption from beri-beri, where it has been noted, has been due to other circumstances than race.

Exposed equally to a common and non-selective cause of infection (and such a cause a miasma or toxic exhalation would be), all races should in all epidemics of beri-beri equally succumb.

But they do not. How, then, explain the separating line between races which does so often actually occur? It is here that the miasma theory breaks down. There can be no such common extrinsic factor of infection. For the causation of beri-beri, with its extraordinary exemptions, we must look elsewhere.

The exclusion of all other possible modes of origin leads us back to the alternative with which we set out—the only possibility remaining—namely, that of a poisoning through food.

the Selangor (Pudoh) Gaol, at the present moment, the convicts alone are attacked; the warders, also natives, but not of the same race, escape. In the Australian pearling fleets, it was noted by Haynes,¹ the 'South Sea men' and the Australian natives were 'entirely proof' against beri-beri, as were also the Europeans. The Malays and Japanese succumbed. In the matter of housing and exposure the conditions were, of course, the same for all the crew.

Finally, as I shall presently show, in the Straits Settlements and on the adjacent Peninsula, throughout the whole area of which the disease is endemic, among a mixture of immigrants of different races, living under precisely the same external conditions, it uniformly spares two—the Indians and Europeans.

The relation of Malays in the Peninsula to the disease furnishes proofs against the possibility of its being due to 'place effect,' which are very numerous. For the opening up of the country has entailed the occupation of innumerable spots all over its area by parties of Chinese; they are engaged both in agriculture and in mining, while there is not a village in which some of them do not squat as hawkers and petty traders. Sometimes the mine or estate is opened up in virgin soil, previously uninhabited jungle; sometimes the intrusion is into areas already occupied and cultivated by Malays. It matters not to the result whether the newcomers have beri-berics among them or not on arrival, or whether they are all (as is usually the case in opening up new mines and estates) healthy men, mostly coolies newly immigrant from China. It equally little affects the issue that the disease may have been wholly unknown in the locality previously. Sooner or later, generally within a few months, beri-beri appears among these Chinese, whatever their locality and whatever their occupation.

The experience of the writer, who has closely studied the conditions of all classes of operatives and settlers in the Native Malay States since 1889, leads him to assert that *this event is invariable*. It would be impossible to discover an instance of any considerable body of Chinese coolies (a score or more) engaged at work in any part of the country for more than six months among whom some cases of beri-beri had not occurred.

It is a commonplace of local observation that beri-beri thus breaks out—'lights up,' as it is said, in 'new foci'—among Chinese, and even imported Malay (Javanese, Boyanese, Bangoerese) occupants of districts, villages, even houses, the previous

¹ *Journal of Tropical Medicine*, March, 1900.

H. Wright¹ has recently made the point the subject of definite experiment in Selangor. On a correct and liberal diet,² at the Pudoeh Gaol in that State, he found convicts yet constantly incurred beri-beri. The writer's own experience confirms these observations.

Or to Bad Food?—Others have from time to time suggested bad food of various sorts, especially damaged, mouldy, or simply decomposed rice, as a cause of the disorder.

Grall, Vincent, and Porée,³ in a report on the terrible epidemics among the labourers recruited from Annam and Japan to work in New Caledonia in 1890 and 1891, sum up their conclusions in the statement 'that beri-beri occurs whenever the coolie is dieted on a *ration coloniale*.' The characteristic distinction of this from European dietaries, they point out, is that in it bread and potatoes are replaced by rice, meat by fish, salt or dry. 'Le beri-beri,' they say, 'est imputable à une altération de l'une ou de l'autre de ces denrées, peut-être de l'une et de l'autre.' 'Everywhere,' they add, 'beri-beri is confined to those who are fed on a fixed diet, which they have neither the time nor the means to alter.'

It was the fact that both the large gangs of labourers (600 Annamese, 700 Japanese) whom these epidemics affected had been dieted at the time, and continued to be so for several months, on rice and fish only. The allowance of rice was a kilo each per diem. The fish was 'salted,' but stinking; there were no vegetables. Immediately fresh rations were secured, in both lots of immigrants the disease at once disappeared. In the case of the Annamese the rice supplied had been distinctly bad and mouldy; that used by the Japanese was sound and sweet. Porée therefore inclined to the view that the really noxious article was the fish.

Or in Particular to Bad Fish?—Many others, from Miura⁴ and Gelpke⁵ downwards, have supposed that fish, dried or salted, and more or less decayed, such as is eaten very generally all over the East, was responsible for beri-beri.

The disease and the habit do not, however, agree in distribution. Such fish is, for instance, eaten all over the interior of

¹ *Loc. cit.*, p. 61, par. 373.

²

Rice.	Meat.	Vegetables.	Beans.	Fat.	Salt and Curry stuffs.
21 OZ.	6 OZ.	7 OZ.	2 OZ.	1 OZ.	1½ OZ.

³ *Arch. de Méd. Nav.*, February-April, 1895, t. 63, Nos. 2, 3, 4

⁴ *Virch. Arch.*, vol. cvi., No. 2, p. 361; vol. cxiv., No. 2, p. 341 (1888).

⁵ *Gen. Tijdschr. v. N. I.*, April 12, 1897, vol. xxxvii., p. 108.

China and India, where beri-beri is rare, and in the Pacific islands, where it is unknown. In Japan, Saneyoshi has pointed out, it is when least fish is eaten (in the summer) that the disease is most prevalent. Dried fish is eaten all over British Malaya by the Malays, who, in their own homes, rarely have beri-beri, and by the imported Indian coolies, who, outside the gaols, never get it.

Pétit showed that fish might be excluded, as well as lack of fat and of nitrogen. He definitely ascribed beri-beri to *bad rice*, and the evidence he offers in favour of this view goes far to prove it.

Finally, the experiment carried out by H. Wright at the Pudooh Prison in Selangor in 1901-1902 is decisive against fish. During a particular eleven months *no fish of any kind was permitted to enter the gaol, and yet numerous cases of beri-beri continued to occur among the inmates*. Fish thus excluded, *there remains only rice to explain the New Caledonia outbreaks*.

Evidence Pointing to Rice as a Main Cause.—Anderson,¹ describing experiences in ships of the Indian Marine Survey between 1894 and 1899, says 'that the cause of the disease was contained in the food was rendered *highly probable*.' But the evidence he offers, in the writer's opinion, is a complete proof of that view. The incriminated food in these cases included *rice*.

Macleod² has narrated an epidemic on board a ship—the *Ancona*—which he holds was 'an experimental demonstration' that the cause of the disease lay in certain articles of food imported from tropical countries. Those he incriminated did not include rice, but examination of his report shows that it was the rice, most likely, which was at fault.

E. Van Dieren, whose work, 'Beri-beri, eene Rijstvergiftiging,' 1897, is a luminous and convincing exposition of this view of the beri-beri question,³ has from the first, and consistently, maintained beri-beri to be due to a poison contained especially in decorticated rice.

Eijkman,⁴ from experiments in fowls, has been led to the same opinion, but believes the poison to be an original attribute of

¹ *Indian Medical Gazette*, September, 1901.

² *British Medical Journal*, 1897, vol. ii., p. 390.

³ I may perhaps add that I had the advantage of reading Van Dieren's able and scholarly handling of the subject in the work named, and his subsequent 'Kantteekeningen' only after the MS. of my own work was closed. The evidence I bring, although entirely independently gathered, and so far as it may be original, must be regarded as a confirmation and extension of Van Dieren's conclusions and results.

Polyncuritis bij Hoenders, etc., 1896.

the rice seed, considered as separate from the whole grain (raw padi), the envelopes of which he believes contain a natural antidote to the seed poison.

Sakaki¹ has adduced evidence to show that rice, innocuous when *newly cleaned*, becomes after some days' exposure poisonous to fowls. He surmises that the change is the result of air-borne saprophytic bacteria which invade and render it poisonous, and that beri-beri in human beings is the result of eating rice altered by like decomposition.

Uchermann,² detailing the findings of a commission appointed by the Norwegian Government to investigate the disease (which is frequent in Norwegian sailing vessels), considers it to be proved that beri-beri is derived from toxins absorbed in food—sometimes rice, often other articles.

The view that the change in rice which ultimately induces beri-beri is due to the production in it of a specific poison, the result of the growth of a specific agent—a parasite or epiphyte, which may at times also naturally attack other grains, a different conception from that of common decomposition—seems to have been long ago more or less vaguely mooted. But I have been able to trace neither the author of this view nor any record of definite evidence in support of it.

Davidson rejected—quite rightly, on the evidence then before him—all the suppositions connecting rice in any way with beri-beri, on the ground that 'no epidemic has been pointed out as corresponding in its distribution with the distribution of particular parcels of rice, as would surely have been discovered if the diseased rice were the cause.' It will be seen that evidence of this character has since been supplied.

In regard to food, therefore, as to other theories of the causation of beri-beri, the experiences which have so far been obtained are contradictory.

But not the Sole Cause.—The result of all these prior observations is that no single article of food has been determined to be constantly the source of the disease.

In regard to one only of them—viz., rice—has it ever been shown with certainty that it has been on any occasion clearly the cause of beri-beri, yet in other cases it has seemed equally certain that it could not have been so. Moreover, by those in whose view rice has been the source of the disease it is as a consequence of *common decomposition* that it has been supposed to have become toxic; but in some of the worst epidemics there

¹ *Sei-i-Kwai*, March 31, April, 30 1903.

² *Indstilling fra Beri-beri-Komiteen, Kristiania*, 1902.

has been clear evidence that there was no such decomposition : the rice was sound and sweet.

A stronger objection is that epidemics of beri-beri have occurred in regard to which it has been asserted that rice has been positively excluded.

Rice alone, therefore, cannot fill the rôle of the sole and effective cause of beri-beri.

But the objections which apply to rice as the single agent do not apply to it as the occasional representative of a larger class.

Seeing that all possible modes of origin of beri-beri other than through food have been definitely excluded, it is certain that some component of the diet of those who incur the disease must be at fault.

This component need not always be specifically the same.

Yet it is, *a priori*, unlikely that the poison, whatever its nature, which causes the malady, should be found in many or very dissimilar articles of food ; otherwise the disease would be almost universal.

If no single specific constituent, at least a particular *class* of food-stuffs may be supposed, therefore, to be involved, and this class must be one the use of which is common both to the Eastern and tropical native who is most affected by beri-beri and the European who in rare epidemics on land, or more frequently at sea, also suffers.

Little difficulty attends the determination what this class of food may be. In the simple ration of the thousands of mining coolies in British Malaya, who pay the heaviest toll to beri-beri, and, indeed, generally in the humble dietary of the natives who suffer most, *meat* rarely finds place. *Fish* of any sort has already been definitely eliminated from the list of possible factors. *Fresh vegetables* are the one thing lacking on shipboard. No *tinned stuffs* are eaten either in the beri-beri-stricken prisons of Malaya or its mines. There remains, therefore, only the class of *cereal food-stuffs*. The cause must be in *grain*.

The cereals eaten all over the world, though not of the same species, are at least all of one tribe. In physical structure they are alike, in chemical composition very similar. In their reactions and mode of decomposition, their liability to decay, to invasion by epiphytes or parasites, to saprophytic growth, they possess features shared, though in varying degree, by all alike. It is, therefore, *probable* that any form of poison ever found in one should sometimes at least be found also in others, and possibly in all.

The determination of the fact that a single species of corn at times carries a poison which is productive of beri-beri would thus afford the strongest of presumptions that the same agent affects at times other grain also.

In the exclusion of all other modes of causation of beri-beri except that of intoxication through food, in the elimination of every kind or class of food except that of cereal grains, we are thus led by rigorous logical process to a conception of the etiology of the disease which is at once capable of harmonizing all the apparent contradictions as to its cause which have been recorded, and to which nothing is opposed *a priori*, or, as will be seen, in experience.

Beri-beri may be accepted as a Grain Intoxication.—Accepting the theory of beri-beri as a grain intoxication, it remains to test its truth as applied to the regions in which the vast majority of all cases of the malady occur.

Connection of Beri-beri with Rice as Staple Food.—Throughout the countries in which beri-beri is endemic—in the Eastern Hemisphere, at least, with its distribution in which only I have direct acquaintance, and with which only I propose now to deal—the staple food of the inhabitants is *rice*. Now, that the area of endemicity of beri-beri corresponded closely with the areas in which rice was grown, that the distribution of epidemics of it agreed generally with the distribution of grain drawn from these areas, that the extent of the incidence of the disease on individuals or groups of them varied within narrow limits directly with their consumption of the grain—all these were facts early observed, and from them the generalization was loosely made that it was to the consumption of rice as such that the disease was due.

At first sight it might seem idle, even absurd, to seek in a habit almost universal the cause of a disease which, after all, affects but few; but for such a state of affairs alcohol seemed to afford an analogy, among the many devotees of which the paralytic victims are rare.

Too much stress could, however, be laid upon some aspects of the relation between rice and beri-beri, as was undoubtedly done by Takaki, Brémaud, and the other upholders of the 'dietetic' theory. This view seems sufficiently met by the single observation that millions in China eat little more than rice, yet beri-beri is not common there—at least, if the evidence of thousands of coolies is to be believed, who yearly migrate to the Malay Peninsula, and make their first acquaintance with the disease—certainly acquire their first attacks of it—in their new abode.

Again, there are some eighty millions or so whose diet is largely—indeed, principally—rice, in Southern India, yet beri-beri, although known in India, is of so infrequent occurrence as to be of no practical importance there. Moreover, the value of the alcoholic analogy has become more dubious than it was, since the Lancashire epidemic has made the conclusion probable that many or most of the cases of peripheral neuritis of the type previously referred everywhere to alcohol are in all likelihood produced by arsenic, or some obscurer poison.

That beri-beri results from mere rice-eating, that rice itself is its *immediate* cause, cannot therefore, as has been said before, be maintained. Yet the observed fact remains, undisprovable, that wherever the disease occurs in the East, there is the rice habit. However idle it may have been to suppose it the direct and only cause of the affection, it were worse than idle to ignore it as an indirect and probable medium. Not, indeed, every native in the East is a rice-eater, but *every beri-beric in the East is a rice-eater*. In my own experience this has invariably been so. In all the records of outbreaks which I have been able to study it has been so. Even in those very epidemics, where observers have sought to ascertain whether the composition of the diet had any influence upon liability to the disorder, rice has never wholly, or for any considerable period of time, been *excluded* from the food of the persons under observation.

The converse of this proposition, moreover, is also true—*persons who do not eat rice do not get beri-beri*.

Some connection between beri-beri and rice, therefore, there undoubtedly is. What is the nature of that relationship?

The explanation now to be given is an induction from the writer's own observations, and those of others—from a body of facts both so numerous in themselves, and spread over a field so wide geographically and in time as to be free from the fallacies attaching to small statistics.

Slowly accumulated, the significance of these observations has but recently been realized.

In this discussion I shall present, first, the conclusion in the form of a theory to be proved later; and secondly, the data from which that conclusion was inevitably drawn.

For the facts will be best appreciated in the light of the theory built upon them.

Action of Grain-parasites.—Rice, it is well known, is grown principally in low flats, where the roots are constantly kept

46 THE CAUSE AND PREVENTION OF BERI-BERI

by irrigation under water. No grain yields more frequent sports, and the varieties distinguished and cultivated by the Malays alone number several hundred. The tropical temperature, and the continual moisture necessary for the rice, afford also the conditions most favourable for other forms of life.

Together with the grain must and do grow hosts of weeds, a microscopic flora—moulds, fungi, bacteria, ferments—the tribes and characters of which remain yet wholly unexplored and undescribed. Many such parasites, mildews and rusts, from time to time appear visibly, and spread over and destroy whole crops. It is not inconceivable, therefore—is, indeed, probable—that some growth—parasite or merely epiphyte, itself poisonous or poison-producing—should occur and spread among the grain, which, invisible to, or at least not yet identified by the eye, imperceptible even to taste, should yet be recognised by that finest of all tests—and for our purpose the crucial one—its effects upon the human economy.

Such a parasite need not invade or infect the ovule or seed, the actual grain. It would be sufficient to be potential for evil did it reside only within the pericarp, or within or upon the glumes forming the investing husk or hull. Dislodged from these envelopes, when the grain was husked or milled, the poison would become almost inseparably mingled, would be distributed, and consumed with the rice. More—from being merely parasitic in its first situation upon the husk—it might survive upon the separated seeds, invading them as a saprophyte when the latter had perished, especially under the conditions of moisture, warmth, and darkness which favour such decay.

At other times the fungus would fall into other media, which it would render more or less poisonous according to their suitability for its growth; or, more commonly, returning to the soil from which it sprang, it would perhaps sporulate, perhaps remain dormant there, until the winds raised it in dust, or it was spread by water at the next irrigation, to infect new crops.

Exactly such a series of events as this attends the growth of the well-known fungus (*Claviceps purpurea*), which poisons rye. The fungus is in this case easily identified forming ergot, a black swollen mass of roots (or mycelium) which replaces the grain. Fallen to the ground, this again germinates producing spores, which are carried by wind or water to their destination.

Resemblance between Beri-beri and other Diseases due to Grain-poisoning. The briefer, intenser onset, the frequent suffering

which follows poisoning by ergot, and the ease with which the diseased grain could be distinguished, made establishment of the chain of its causation comparatively simple. Yet it was centuries before that cause gained recognition.¹

In pellagra another illustration is afforded of a disease signalized by the presence of grave nutritional and nervous disturbances which is produced by the consumption of diseased grain.

The malady, endemic in Italy, Hungary, Roumania, Malta, Egypt, is slow and chronic, the time required for its establishment in its victims extending over years. In this case, the maize, the medium in which the intoxicating agent is produced, may or may not present obvious signs of alteration or decay. But neither any toxin nor the specific organism producing it has so far been identified.²

Now, apart from the symptoms, there is no disease which presents a closer resemblance to ergotism in the general facts of its epidemiology than does beri-beri.

The insidiousness, the insignificance of the earliest symptoms, the prolonged period of 'incubation' and onset of the latter disease, above all, the complete absence of any evidence indicating correspondence between its epidemics and the condition, distribution, or consumption of particular crops or consignments of grain, have so far militated against the demonstration for it of a morbid cause similar to that which has been shown to obtain in the other grain intoxicants. But some such agency, the train of reasoning which we have followed indicates, there must be.

Let it be assumed, then, that it is so—that beri-beri is caused by a poison conveyed in rice; that this poison is generated in it by some rust, fungus, microbe, ferment, mildew, mould, often or commonly growing on and gathered with the crop, able possibly also to continue to multiply in it when stored, and especially when damp or decayed.

It is to be observed here that rice is postulated only as being the *common or usual*, but not a *necessary*, habitat or medium for conveying the poison. Beri-beri, as the result of the operation of the same or a similar agent, in other articles of

¹ Thuillier first proved conclusively the relationship of ergot to its gangrenous and other effects, by feeding animals upon it, which developed these disorders, in 1630.

² Another disease probably owning similar etiology is Strachan's disease, occurring among natives in the West Indies, which in some respects resembles pellagra. The West Indians live largely, too, on maize.

48 THE CAUSE AND PREVENTION OF BERI-BERI

food or other cereals, may very well occur under circumstances in which rice could not play a part. Such cases, from the present point of view, would be exceptions, which would not, however, militate against the truth of the proposition now under discussion.

Corollaries of the Rice-poison Theory.—Among corollaries of the theory would be :

1. That *rice grown in a country in which the cause of disease was not endemic¹ would be incapable of causing beri-beri*, and that, so long as it was preserved from contamination, it would remain so, even in a locality where the disorder was endemic.

2. That *grain proved to be non-toxic in one place might become toxic in another*.

3. That *grain kept in the same place and non-toxic at one time might become toxic later*.

These last two conclusions flow from the conception that the primary morbid agent is an organism capable of reproduction. For, although there may have been none of the presumed 'fungus' (as, merely for convenience, it may be agreed to designate the toxin-producer) present on the grain when harvested, yet in a region throughout which it is, by our premises, widely distributed it is more than possible that stock may become contaminated later, whether by air-borne spores or through contact with things themselves already infected, as from storing in dirty bins. This would apply not only to unhusked, but also, perhaps more, to hulled grain, and even, it is conceivable, to rice ready cooked for food.²

¹ It may be doubted if there is any such place.

² When the reflection is made, how rapidly masses of organic matter—*e.g.*, food-stuffs—change, decay, and often become poisonous, under the influence of appropriate ferments—how starch, for instance, during its short sojourn in the mouth, is turned by ptyalin into sugar; insoluble albumins by pepsin into soluble albumoses and peptones, which are poisonous if injected directly into the circulation—it is far from inconceivable that a mass of soft, cohering, starchy matter, such as cooked rice presents, should be converted in part by the action of some such agent as has been postulated into compounds as distinct as alcohol and yet more poisonous, and this, perhaps, in a period of time which might be reckoned, not by hours, but by minutes.

In the vegetable world such ferments are widely spread, and have been closely studied. The most familiar among them is the diastatic ferment of barley-malt. It is intimately attached to the grain itself, the starch of which is, through its action, converted into dextrin. This ferment is not killed by drying; but when the ground malt is placed in water, the diastatic action of the ferment begins, and continues until stopped by boiling the grain. From the

4. *All rice derived from beri-beric areas must be regarded as potentially toxic,*¹ although absence of ocular demonstration of the fungus, or of direct identification of the poison renders it impossible at present to distinguish toxic from non-toxic samples, otherwise than by their pathological effects.

5. *The extent to which given parcels of rice are affected will be variable.*

It is conceivable, though hardly probable, that the whole of a season's crop from a given district should be poisonous. On the other hand, only a few heads of the grain here and there may be so. The derived rice, as sold commercially, being the result of admixture of many samples from different localities, will average these differences; but it will always remain a possibility that different portions of one crop or cargo, even perhaps of one particular bag of grain, may exhibit differences in toxicity.

The degree of toxicity of affected rice will depend :

(a) Upon the quantity of poison originally present in the grain. This will depend on local (meteorological) and other conditions affecting the harvest.

(b) Upon the quantity of the poison which may have become mixed with the rice when the envelopes were separated from it in milling.

(c) The time which has elapsed after hulling, during which the poison (fungus) shed into the rice (if a saprophyte) may have been able to multiply—the length of time, in fact, for which the grain or rice may have been stored.

dextrose and allied bodies in such fluids (the 'worts') alcohols are evolved through the agency of moulds (or yeasts).

Another and apter example of such ferments is yielded by a fungus (*Aspergillus oryzae*) which grows on rice itself. A small quantity of this body, 'taka-diastase,' added to a mass of starch suspended in water, will convert it into a clear solution of dextrin in a few seconds.

The ordinary boiling-point temperature will not destroy every spore, and in large masses of rice which are being slowly cooked even this temperature does not necessarily obtain. But water, the essential for fungoid growth, is thereby added, and it is possible that, upon cooling, spores which the heating has been insufficient to destroy should immediately germinate, or ferments be liberated the effect of which should be to produce a toxin in the rice with the same rapidity as happens in the fermentations mentioned above.

The state of affairs here conceived is, however, only academically postulated, since in relation to beri-beri the facts are, as will be seen later, susceptible of an easier interpretation.

¹ *I.e.*, as likely to hold the poison, which will produce its effects when the rice is used elsewhere; only the event can show if the rice is actually toxic.

(d) The favourableness of the external conditions for the spread in the raw grain, or the rice, of a parasitic or saprophytic growth. Principal among these would be moisture, darkness, warmth, and perhaps natural death (or loss of germinating power) in the seed.

(e) The intensity of the poison may also vary with the degree (if it is due to parasite or epiphyte) of its maturity when gathered, as in the case of ergot.

(f) Influences, such as age, exposure to air, light, and desiccation, which, in the case of ergot again, are known to diminish its power.

(g) The *strain* or particular virulence of the actual agent in the actual parcel of grain concerned.

6. *The poison*—organized body, fungus, with any soluble ferments or alkaloids already formed—so far as it causes visible differences in the affected grains or on envelopes, or affects only the surface of the seed, *should be separable by appropriate methods from the rice.*

Thus visibly affected grain might be sorted by hand-picking. Again, at an early period after gathering, before the parasite has had time, or been placed under conditions enabling it to grow and invade the ovule—possibly at any period in grain kept since harvesting perfectly dry and under conditions preventing multiplication—it might be possible so to treat the grain by washing and soaking, boiling and drying, either before milling or after, that all adherent poisons should be washed away, while spores or other organisms, first induced by the soaking in water to germinate, would in that, the most vulnerable stage of all such organisms, be killed by the subsequent boiling. In fact, by such means a perfectly clean and even efficiently sterilized rice—‘cured rice’—would be turned out for consumption.

Such rice, free from contamination at the outset, if properly stored, being above all kept dry, and not contaminated by admixture with other possibly affected grain, should ‘keep’ indefinitely, and remain permanently wholesome, and incapable of causing beri-beri in its consumers.

In grain, however, of which the whole element should originally have been invaded and become toxic, or in which, through age or exposure to damp, the fungus had extended like a saprophyte into the actual body of the seed, such separation of the poison could not be effected; nor need heat nor even boiling alter it.

7. The original source of the poison being something organic,

and its production liable to be conditioned by meteorological factors, *toxic rice will tend to be confined to limited geographical areas*. Its abundance in crops will be influenced by local conditions. But roughly, no doubt, the distribution of the fungus would correspond with that of rice, conditions suitable for the one being generally favourable to the other.

8. Lastly, so far as the poison may be due to an epiphyte or saprophyte which can grow on rice, or to which rice is peculiarly liable, *it will be liable to be conveyed to all countries to which affected rice may be distributed*, and will thus be likely to form new 'foci' of endemicity by infecting the local grain.

Characteristics of the Disease Deducible from the Foregoing Data.—Applying now to the *epidemiology* of the disease the conclusions properly deducible from these data, it becomes predicable in every epidemic of beri-beri that :

1. *Those whom it attacks should be rice-eaters.*
2. *It should not attack those who do not eat rice.*
3. *Among rice-eaters its incidence should vary with the sort or quality of rice eaten.*

The incidence of the disease upon eaters of *affected rice* should vary, moreover, with the extent to which the processes to which the grain was subjected before use could be conceived as capable of modifying or destroying any organized agent or soluble products of it attached to the husks and pericarp. It should be thus, if the poison be epiphyte or saprophyte, or both :

(a) Most frequent amongst those consuming old rice prepared by directly hulling the grain, without any preliminary washing, sorting, or heating.

(b) Less frequent among those who, preparing their own rice directly from the grain in small quantities as required for daily use, are wont to carefully separate unsound grains from the mass, and also to *thoroughly wash* the grains of rice free from all adherent matter before cooking and eating it.

If the poison be merely saprophytic :

(c) It should not occur at all among users of rice which should have been submitted before hulling to any process which could be regarded as sufficient to properly cleanse and sterilize it.

4. Among eaters of any sort of rice which the event shows is or may be poisonous to some, other things being equal, *the case-incidence* (or number of persons attacked) *and the case-mortality* (the severity of incidence upon individuals attacked) *should vary directly with the quantity of rice eaten*—

(a) Absolutely.

(b) Relatively to the quantity of other articles composing the diet, as a poison taken alone will often produce a severer or more rapid effect than the same or even a larger dose of it taken together with other food.

5. *The course of the disease in those affected should not tend, or tend little, to recovery*, and the extent of particular epidemics—i.e., the number of persons attacked in a given community, when all are not affected simultaneously—should persistingly increase so long as they continue to use the same rice which they ate when it was acquired; and, conversely, it should so tend, if not invariably, at least more often than not, to recovery when the noxious rice is discontinued.

It is the truth indicated in this obvious deduction—one impossible to be made from any other than the rice theory—which is involved in and explains the frequent seeming connection of beri-beri with *places*. For it is not place, but one of the accidents of place—namely, the food-supply—to which the effect should be attributed, although, for want of complete information, the operation of the latter factor has often been lost sight of, or been confused with the other.

6. Following from the preceding, and quite apart from fluctuations associated with geographical distribution of the prime agent (the fungus which poisons the grain, the true 'endemic' factor, which annual and secular mutations may affect), *beri-beri will therefore appear to be conditioned by locality*, to be a disease attaching to, and persisting in places (houses and ships) in a manner apparently (apart from our theory) inexplicable through any other conception than that of a *contagium* planted permanently in them. For it must happen (according to the view of its causation which has been postulated) that, as the distribution of cases follows exactly the poisoned food-supply, certain places or institutions should suffer alone in the midst of others which are free. The abrupt disappearance of the disease from places in which it was once prevalent, and in which the outward conditions have in no way altered, will result from the same conditions.

7. As depending upon two variables (the toxicity of the actual sample of rice in consumption, and the susceptibility of the individual concerned), the '*latent*' period, or length of the term of exposure of persons to the action of the poison before they show effects, *will have the utmost irregularity*, both in different

epidemics, and in different individuals in one and the same epidemic, to an extent not to be paralleled in diseases due to contagia, or at least those in which the action of such a factor is definitely known.

8. So far as the term 'endemic' may be held to imply that the occurrence of the disorder to which it is applied is conditional upon the presence of particular extrinsic factors peculiar to the locality in which it occurs, and without which it cannot occur, *beri-beri will show no true endemicity. It will develop equally in any part of the world to which poisonous rice can be carried, and among all communities (whatever their race) who consume it.*

It will be easily transportable, be spread by traffic and human intercourse, be introduced and even appear, like an infection, to be multiplied through such agencies, in places where it was previously unknown.

9. On the other hand, *it will clearly not be incurred through mere association (contact) with persons affected*, and so will afford no data of multiplication anywhere through contagion, supposed instances to the contrary being readily explicable through factors already cited.

10. As dependent merely upon the supply of the intoxicant, *epidemics of beri-beri will be liable both to begin and terminate abruptly*, without any appreciable change in any extrinsic factor, other than alteration in the food-supply, to account for such cessation.

11. *No constant relation should be determinable in epidemics of beri-beri between the prevalence of cases and external (meteorological or telluric) conditions coinciding with them.*

For not only must the principal factor, the original infection of the grain (or its subsequent toxification by added fungus), have been, usually long, anterior to its consumption, but the disease itself (as conceived) is the accumulated result of successive small doses of poison, the absorption of which must have been spread over a considerable period.

12. The product, rice, being in itself a seasonal one—only one crop is harvested annually in the countries in which it is principally grown¹—*apparent seasonal variations in epidemics may occur*, modified by the interval which elapses in countries using imported grain between the finishing of the former year's stock and its replacement by the new year's grain. Epidemics

¹ *I.e.*, Cochin, Siam, and Burmah. In India there is both an autumn and a winter crop raised.

54 THE CAUSE AND PREVENTION OF BERI-BERI

so dependent on particular supplies will not begin at once upon the delivery of new stock, but only after they have been in consumption for some time. That time will be equal to the interval, or latent period, necessary for poisoning to result from consumption of the particular sample of rice concerned, and may be of any duration, but usually some six or more months; so that autumnal outbreaks would result from crops which came into consumption, for instance, in the preceding spring.

13. On the other hand, the original infection of the grain being modified by meteorological factors, *annual, and possibly multi-annual, or secular variations in the prevalence and severity of epidemics are to be expected.*

14. Under certain limited conditions, strictly definable, as in the case of communities settled permanently in one district, and subsisting on rice grown locally sufficient for their own consumption, through which beri-beri should be incurred, correspondence might be noticeable between outbreaks and closely antecedent meteorological conditions, as heavy rainfall, blighted harvests, etc.

15. *Epidemics of beri-beri should tend to occur oftenest under circumstances which necessitate the prolonged keeping of rice in closed and dark receptacles (such as ships' holds) and the consumption of stale stock, more especially when to these were added other conditions, such as moisture and warmth, which would favour either first infection by fungus or proliferation of fungus by which it was already infected. Epidemics will thus appear, and be most severe in places most remote, in point of transport facilities, from sources of food-supply; and conversely beri-beri will become more widespread but less fatal as these facilities increase.* For the first condition involves greater exposure to influences damaging to the grain, as prolonged storage, and the accumulative effect of constantly consuming rice increasingly poisonous; while under the latter it becomes easy to renew stocks which, thus stored for shorter periods, are less decayed. Moreover, all parcels of rice not being equally or necessarily at all poisonous, the chance of continued use of an infected sample is proportionately reduced.

16. For reasons similar to those which determine the considerations given above, *beri-beri should be apt to affect most often and extensively public institutions in which many persons are housed together*, for, although it may not be pleasant to confess, it is idle to ignore the fact that it is precisely in such institutions

that the factors which make for the provision of a cheap, and therefore probably bad, food-supply have most play. It is an object to run all such establishments economically, and in prisons and asylums, schools and orphanages (in the East, at least), the food—the rice—is supplied on contract, offered for tender, when, if not always the lowest, certainly not the highest offer is accepted. It thus becomes an object with the contractor, who has to supply rice at a fixed, and sometimes actually losing, rate, to put in the cheapest article which can pass inspection.

It is to be remembered that it is no ordinary mouldiness in rice which is, according to the theory, responsible for producing beri-beri. It is due to nothing that can with our present knowledge be detected at sight, so that these remarks cannot be held to convey the slightest imputation upon those whose duty it may be anywhere to perform such inspection.

The same considerations apply to the provisioning of ships, the rice supplied to the native, and sometimes shared by the European crews of which is probably often bad.

To the above deductions, applying to the general epidemiology of the disease, the following may be added as applicable to what are called its 'clinical' manifestations :

17. As a complaint not due to the multiplication in the system of a living germ, the effect of which is to produce in most of such diseases an active immunity against subsequent attacks, but as dependent merely on the absorption of a definite (fairly stable) chemical body, no true immunity, the analogy of all intoxications renders it permissible to suppose, will be conferred by one against a succeeding attack of beri-beri. It will not 'protect against itself.' Some degree of toleration for, or habituation to, the effects of the poison—'passive immunity'—it is to be surmised will, as in the case of many other chronic intoxications, be attained, increasing perhaps proportionately with each increase of dose short of that producing an actual attack; but complete immunity, even of the 'passive' order, or of more than fleeting duration, will never be conferred. There will be no period, that is to say, even immediately after recovery from a severe attack, during which the further application of the cause will not, if sufficiently strong, produce further effects.

Rather, it follows from the same conceptions that beri-berics should be particularly liable to remissions or relapses, this being no more than an expression of the fact that, where no true immunity against the effects of a poison is conferred, the condition

56 THE CAUSE AND PREVENTION OF BERI-BERI

entailed by it will recur again and again as often as the excitant is applied, while each recurrence marks the breakdown to some extent of the protective factors within the organism, and diminishes the total efficiency of its resistance to the disease.

18. The clinical course of an attack, or of different attacks of beri-beri in one and the same individual will be, therefore, like its incidence upon the community, subject to the utmost irregularity.

Each attack may be mild or severe, interrupted or continuous ; it may last but for hours, or it may last for years.

19. Dependent partly upon the resistance of the individual, partly upon the relative toxicity and irregularity of consumption of varying samples of rice, there will be variations in the degrees of severity with which the disease attacks different persons, even in one and the same epidemic, which are to be paralleled by the records of other accidental and food poisonings, but not by the history of any specific disease.

Thus, in a limited community, between whose members no difference in race or sex, little in age or physique, and nothing in occupation or any circumstance of environment can be discerned, some will succumb (apparently immediately) to the swift fulminant or pernicious form, others exhibit only the mildest manifestations of the disorder.

Moreover, the severity of the initial illness will afford no clue to the issue, either as to fatality or speed of recovery. One whose signs are at the onset but trifling may die after a few hours, and without warning of the event ; another whose symptoms are of the worst may incline to speedy recovery. All this is to be predicated of the operation of a poison, subtle, variable, and broadspread, consumed irregularly and at random, at moments and in quantities which cannot be calculated because the actual agent is as yet unknown.

Such, with some of its corollaries, being the theory of the causation of beri-beri through rice, it remains to show that it agrees with fact.

For the logical proof of any theory it is necessary to show not only that all the inferences which can be properly made from it are verifiable, so far as they can be put to the test of experience, but also that some at least of the facts which constitute that experience are incapable of explanation on any other theory.

Many among the epidemiological and even clinical features of beri-beri predicated in the above postulated mode of its causation

might equally well have been deduced from any other theory. With the exception of the first five, they might all have followed from almost any kind of poisoning conveyed in food, while some would accord well enough with the conception of the disease being due to a living parasite.

It would be superfluous to examine deductions of this kind in detail. Those who are well acquainted with the natural history of beri-beri will recognise that they represent nothing more than a picture of the most familiar features of the disease.

The first five of the deductions are, however, of a different kind. They are such as could obviously be made from no other premises than those of the rice theory, as it has been formulated here. The pivot upon which the application of each one of them turns is rice.

It is by these, the peculiar predicates of the rice theory, that the latter itself must stand or fall.

The ample array of facts by which they are demonstrated form the principal part of this work.

Before discussing them, however, a matter must be dealt with as to which there is division of opinion, but about which a clear understanding is essential for critical appreciation of the conditions under which beri-beri is actually produced.

This is the question of the 'incubation' or 'latent period.'

SECTION III

LATENT PERIOD

THE 'LATENT' OR 'INCUBATION PERIOD' OF BERI-BERI, AND THE IMPORTANCE OF ITS BEARING UPON EXPERIMENTS

Etiological Importance of Latent Period.—The interval elapsing between the inoculation, in infectious diseases, of the germ, or the first reception, in intoxications, of the poison into the system, their effects upon which constitute the disease, and the moment when the injurious results appear—the 'incubation' or, as in intoxications it may better be called, the 'latent period' of the disorder—is obviously of especial importance in attempting to discover the cause of a malady such as beri-beri, the common source of cases of which, in its epidemics, no incidents of contagion afford us ready data to trace.

Narrow Limits of its Variation in Infectious Diseases.—In the acute infectious diseases of which the specific causal agent has been determined (and not less so probably in those the natural history of which makes it all but certain that they must be due to similar living organisms), the duration of the incubation period depends, it may be shown experimentally, somewhat upon the initial dose or quantity of germs introduced, but more upon their vitality (the speed with which they multiply), this again varying inversely as the resistance of the subject, or, in other words, the unsuitability of the conditions within the body for their development.

The intensity of the illness, being in proportion to the poisons elaborated, depends chiefly, it would seem, upon prior conditions, external to the patient, which have effect in increasing or diminishing the virulence of the strain of germ—*i.e.*, its capacity for producing in different degree or intensity those poisons through which the essential morbid effect in the system is produced. But in such diseases, whether the illness be mild or

severe, the period, the whole course of their evolution, remains, for specific complaints, very much the same. The variation from the normal, for particular cases, is never very large. Such differences in their course as do occur, in point of time, are to be measured almost always by days, rarely by weeks, never by months. This applies especially to the incubation period, which appears never to be extended more than a few days in any of the diseases in which acute illness may supervene a few days after the introduction of the virus.

A latent period of more than a month is not claimed, on good authority, for any one of the acute general infections. In the majority a fortnight is assigned as covering the limits of the period of incubation.

Its Great Irregularity in those Due to Poisons.—In diseases produced by poisons introduced as such, the result manifestly depends chiefly upon quantity. Illness may immediately follow a single sufficient dose, or may only ensue as the result of an accumulation of repeated small effects. In such cases there will be the utmost irregularity in the latent period, which may thus be anything between days and years.

This Irregularity characterizes Beri-beri.—In the causation of beri-beri, as postulated by the rice theory, it is not merely the quality of the rice eaten, the degree to which it is toxic in the single doses taken at each meal, which is important. It is, usually, the accumulative effect of the long-continued consumption, the repeated assimilation of the poison in small quantities taken daily for a prolonged period, which, like that ascribed to alcohol, or small quantities of arsenic in beer, ultimately produces paralysis. It thus, as already predicated, should have no incubation period, no fixed or definite term of production. The duration of time for which it might be possible for the poison to be absorbed before the production of symptoms must be always irregular and often prolonged.

The determination of the inferior limit of the incubation period in any disease is easy. It is merely the shortest interval which has been noted in any case between the beginning of illness and a single possible exposure to the cause.

The maximum period in the case of diseases the cause of which is certainly known and is some parasite only found in them, can also be determined by data of contact.

But when such a cause is ectanthropic, some saprophyte only facultatively parasitic in the human being—still more so when the morbid agent is quite unknown—determination of the limits of

incubation can be but approximate, and the ways of making them but two: either a period can be assumed, and tried against the facts, or, the circumstances of a sufficiently large number of cases having been investigated, the induction may be made from them.

Could a number of susceptible subjects be placed under observation, under external conditions made in every respect equal for all, in the known presence, or under the possible exciting cause of the disease¹ (as declared by its manifestation in any of them), and for a period sufficiently prolonged to cover the possible outside limit sought, the result might, for all practical purposes, be relied upon as affording valid conclusions.

The period at which the greatest number of cases became ill would represent the average period of incubation of the disease due to the cause under those circumstances, the latest period at which any subjects succumbed the maximum limit of its duration.

In the case of beri-beri it is never possible to say for certain when exposure to the possible cause began. Even if, as supposed, the cause be *rice*, it is not possible to tell, even, how long its toxic effect has been in development without culminating in symptoms. But the *shortest* probable limit of the latent period would appear to be determined by some observations of Anderson's made in epidemics occurring upon ships of the Indian Marine Survey during several consecutive years (*Indian Medical Gazette*, September, 1901).

'On October 17, 1894,' Anderson says, 'the *Investigator*, with about 120 people, and the *Nancowry*, with about twenty-six people on board, left Bombay for Karachi, to survey the mouths of the Indus. On October 21 one of the *Nancowry's* quartermasters became affected with a slight fever, reaching a little over 100° F., but subsiding in a few days to normal, cedema of the feet and legs, which subsequently spread to the abdominal wall, tenderness of the calves, great weakness of the lower extremities, and pallor.' The disease was diagnosed as beri-beri.

'As he showed no signs of recovering, he was discharged to his home on November 2.'

If beri-beri does not occur in Bombay,² if there was no possibility that this patient could have been already partially affected, in the state which I shall show later must be conceived to be comparatively common and may be provisionally termed the

¹ *I.e.*, exposed for the first time, or after an interval of entire freedom from any possibility of incurring the disease.

² It is not endemic in Bombay, and has not been epidemic there for many years.

sub-beri-beric condition, then the incubation or latent period of beri-beri in this case could have been no more than *four days*.

Other cases continued to occur on both ships at quite irregular intervals. The second happened on the *Investigator* on November 7 (twenty days), another on November 30 (thirty-three days), and thereafter 'one or two cases were almost daily reported until the middle of December.'

In 1896 'the first case occurred within *a few days* of leaving Bombay,' in a Goanese cook. 'The following day a lascar was affected, and then a lull occurred until December 12, when a Goanese sweeper reported himself sick. Next day a stoker, and on the 17th and 18th two more stokers, were taken ill. Thereafter the disease ceased.' In 1896 there were four cases on one ship; in 1897 only two cases on the same ship.

In 1898 'only one case occurred, about a fortnight after leaving Bombay. . . . Since then the ship has been free from the disease.'

There is no reason to doubt these being ordinary cases of beri-beri. Whatever the cause of it, it was present on the *Nancowry*.

The crew, it is to be observed, must all of them have been exposed to its presence, and to equal chances of infection—were the cause infectious—simultaneously. The first case showed that the disease was a rapid one, since the whole train of changes was produced within a week. Yet, while one subject succumbed immediately, others did not do so. Most of the cases took a month.

The bearing which other facts in this highly interesting report of Anderson's have upon the etiology of beri-beri will be discussed later. I only quote it here as fixing probably the earliest, or an extremely early, period of incubation for beri-beri, and for the sake of insisting upon what is of the greatest importance—the fact that even in epidemics in which that period is shown to be in some cases as short as four, in others, who are by the conditions equally exposed, it may be as much as sixty days.

In an epidemic described by Joynt occurring among Japanese coolies imported into Fiji in 1894, the first cases occurred within two months of arrival on the island, two and a half after leaving Japan. The coolies were all selected, healthy adults, free from beri-beri on leaving Japan. The disease was previously unknown upon the island. The circumstances determining it must have begun, therefore, with the embarkation. The disease progressed until, by the end of six months, all but eight out of fifty coolies composing one gang, and 226 out of 250 composing another, had been attacked, when the survivors were sent back home.

Here the conditions make it almost certain that, whatever the

causes of the malady, all the coolies were exposed equally to them. The latent period, however, varied much among individuals. But, from whatever date the first 'infection' may be reckoned in this epidemic, it is clear that in the majority of cases the latent period was at least two, while in many cases it was more probably three or four, months (see *Journal of Tropical Medicine*, May 1, 1901).

In the remarkable outbreak described by MacLeod¹ occurring on a sailing ship—the *Ancona*—which attacked only the officers, and no member of the crew, the ship left a beri-beri-free port (New York), bound for Shanghai, on August 19, 1896. The disease was noticed by the first patient at the end of December. Symptoms began in the other (four) cases a few days later. There was no possibility of infection having been brought upon this ship at any intermediate place or time, as she touched nowhere in the interval. The latent period in this instance would seem to have been the same in each of the five cases—about five months for a maximum.

Other instances to the point are supplied in the account by Haynes of epidemics occurring in the Cossack pearling fleet (*Journal of Tropical Medicine*, March, 1900). Haynes gives notes of six voyages, upon all of which, except two, beri-beri broke out among the crew.

The cases which happened appear to have been generally severe and rapid, some of them pernicious. The crews dealt with were of mixed races, but mostly Malays, and all originally selected for their physique and freedom from disease. The disease began on the first ship during a seven months' cruise, on the first cruise of a second ship in the fourth month, on the same ship during a second cruise in the third month, and during the last two of three more cruises during the seventh month. The only voyage upon which no beri-beri at all appeared was the fourth, during which 'a mixed diet was adopted.' 'It had by this time been agreed that every endeavour should be made to return each batch of men within seven months, and on acting on this stipulation during No. 4 cruise the result was no appearance of beri-beri at all.'

'The fifth batch were also returned home safely in the seventh month, although the bulk of the rice used during the earlier part of the cruise had suffered by sea-water. During the last month two men showed symptoms of the disease, but reached home safely, aided by a liberal supply of mutton, potatoes, and beer.'

¹ *Loc. cit.*

'The sixth cruise lasted nine months, from November, 1886, to August 18, 1887. In April the stores were again damaged by sea-water, but eventually replaced. On the morning of June 9, the seventh month, a man was taken ill with the disease, showing all the symptoms in its rapid form, with swelling of one leg. He died at 7 p.m.' A month later 'it was decided to return ten men who had dropsical symptoms. . . .' The relation of rice to these cases will be dealt with later on, but Haynes's conclusion, 'that with proper care it [beri-beri] will not develop in less than seven months,' commends itself as fully justified by the record given.

When a crew of some eighty to a hundred natives is confined, as was the case here, upon a small schooner, it would seem impossible either that any of them should escape any infection, the influence of any cause of disease present, or that, such an agent being present, as by the conditions it must have been, in these ships from the time of embarkation, its operation should not have begun upon all of the crew from the same date.

The inference is again made clear, from the facts related above, that the condition or conditions which produce beri-beri, if in some cases they produce disease almost immediately, in others do not do so until they have been in operation for several, usually many, months.

Upon twenty-one vessels which brought cases among their European and native crews into British ports between September, 1901, and October, 1902, the circumstances of which were inquired into by the Commission on Arsenic-poisoning, the stated periods of possible exposure to the poison on the ships varied between fourteen days and eighteen weeks, and were in no two alike. Bullmore¹ gives a similar series of determinations in regard to forty-three ships bringing beri-berics to Falmouth between 1896 and 1898. The voyages varied between fifty and 195 days.

In Java, Pekelharing says it is, as a rule, not until after one or two years' residence in an endemic area that Europeans, newly arriving, contract beri-beri.

'Hoffman says that "strangers do not take the disease until the second year." Christie, Hunter, and Aitken state that it only develops after a stay of from eight to ten months in a place where beri-beri prevails.'² Pekelharing describes cases among native soldiery which appeared to have developed in, at the

¹ *Lancet*, September 22, 1900, p. 873, and *Public Health*, January, 1903.

² Pekelharing et Winkler *Recherches*, etc., p. 15.

shortest, five weeks. But such rapid cases are, I shall show later, in most instances probably but apparent.

Scheube,¹ speaking from his own experience, says that 'immigrants only become affected after a long stay in a beri-beri district. . . . The time that elapses between the period of immigration and acclimatization varies. . . . It fluctuates between weeks, months, and years. A period of over six months is, however, the rule. . . .'

Hirsch² states 'the predisposition to an attack of beri-beri increases with the length of time spent at the focus of the disease.' He cites Calhoun as proving this for Ceylon, Hamilton and Malcolmson in India, Simmons in Japan. He quotes Baelz as saying that the period required for becoming predisposed to the sickness is to be estimated at from a few months to a year. 'The same period is fixed by practitioners in Brazil, where strangers are all exempt from attacks of beri-beri for six to twelve months after they have come to reside at one of the endemic centres of the disease.'

At an unusually severe centre of the disease, Christmas Islands—nearly every resident upon which seems to have had beri-beri—Durham found increased knee-jerks in two men who had only been sixteen days on the island, and had been passed as healthy before arrival (at Singapore). One of these died forty-four days later. Of 100 consecutive cases of fatal beri-beri, presumed to have arrived healthy, the deaths took place at irregular intervals after arrival on the island—11 per cent. after two, 21 per cent. after three, 6 per cent. after four, 12 per cent. after five months' residence. Up to six months 57 per cent., six to twelve months 21 per cent. One-fifth of the cases had been twelve to eighteen months in residence, and only two over that period.

In the Malay Peninsula, it might be supposed the thousands of cases annually occurring would afford ample material for determination of the 'incubation' period. Most of the patients there are Chinese coolies, imported into the country to work under contract for a term of years (one or two) at mines or upon estates. These men are called in their vernacular 'sinkhehs'—*i.e.*, 'new-comers.' The earliest dates at which such sinkhehs should succumb would mark, of course, the shortest incubation, since beri-beri does not occur in their own country. But the difficulties occur: first, that the accounts of these men are hardly ever reliable as to time. Their calendar in no way corresponds

¹ *Krankheiten der Warmen Länder.*

² *Historical Geographical Pathology*, vol. ii., p. 573.

with ours, having no weeks. They are quite unobservant, and take no account of a few days more or less. Secondly, there is the difficulty of eliminating the factor how far they may have been exposed to the influence, whatever it is, which causes the disease, after their engagement, and before their arrival. For they undergo detention, first, at the ports in China at which they embark, in most of which beri-beri occurs; secondly, on the passage to the Straits Settlements, which takes two or three weeks—often, in the smaller craft, much more; and thirdly, in dépôt in Singapore—a stronghold of beri-beri—for never less than a week, frequently two or three weeks, before they finally arrive at the locality in which they are to labour. As a matter of fact, while a few of the hospital cases assert their disease to have been acquired within a week or so of arrival here, the large majority give account of much longer exposure. Many of my cases are old hands, who have been in the country for one or two years before getting the disorder.

H. Wright,¹ interrogating 276 sinkhehs who acquired beri-beri for the first time shortly after arriving at the mines—'infected centres'—in Selangor at which they were imported to work, obtained the following results:

		Number of Cases acquiring Beri-beri after										
		10 days.	14 days.	30 days.	2 ms.	3 ms.	4 ms.	5 ms.	6 ms.	7 ms.	8 ms.	Total.
DATE OF ARRIVAL.		ARRIVALS IN S.W. MONSOON.										
First month of S.W. Monsoon, Apr.	{	— (April to May.)		1 (June)	3 (July)	2 (Aug.)	20 (Sept.)	32 (Oct.)	43 (Nov.)	41 (Dec.)	142	
First month of N.E. Monsoon, Sept.		ARRIVALS IN N.E. MONSOON.										
{		12 (Sept. to Oct.)	32	34	42 (Nov. to Dec.)	— (Jan.)	14 (Feb.)	— (Mar.)	— (Apr.)	— (May)	134	

Accepting the statements of these coolies as reliable, and assuming that the cause which determined the disease began to operate only from the date of their arrival at the mines—the 'infected centres'—the inference is correctly drawn by Wright that the latent period was, in the case of some of these coolies, as short as ten days.

¹ 'Study,' No. 1, p. 11.

But it is no less plain that in the majority it may have been, and probably was, much more. Wright says that the fact that 'no less than twelve' (out of 276) coolies acquired beri-beri within ten days of arriving at an 'infected focus' 'establishes beyond dispute that the incubation period of beri-beri is not longer than that of other infectious diseases.' The statement is not precise, but if the writer mean, as he seems to do, that the incubation period of all cases of beri-beri is no more than ten days, his conclusion is certainly against the probabilities, on his own evidence. The reference to 'infected foci,' the comparison with 'other infectious diseases,' are, of course, assumptious in Wright's argument—begging the very question which this determination of the incubation period is sought to prove.

But there are few infectious diseases—none producing rapid acute illness—of which the incubation period has been shown to be as much as a month, whereas the date at which 75 per cent. of Wright's cases got the disease exceeded that.

The more important conclusion to be drawn from Wright's table is that the latent period of beri-beri varies very greatly in different individuals, a result in harmony with other observations. Wright attributes the difference in the dates at which beri-beri became incident on the patients in these two series of cases to some factor connected with the change of monsoons, and he supports the view by another series (of ninety cases) occurring under his observation in the Pudooh Gaol (May, 1901, to March, 1902). But that there can be little in this argument another series of cases proves, observed by Travers¹ in the same gaol during 1902, in which precisely the opposite relation to the monsoons occurred.

Travers' series comprised 478 'new' cases, of which 291 occurred between the beginning of March and the end of August (the south-west or so-called 'drier' monsoon), and only 184 during the six 'wetter' monsoon months of September to February. The rainfall in the latter period was 71.67 inches, and in the former 44.73 inches. It is to be observed that seasonal periodicity can hardly be invoked to explain the differing incidence presented by Wright's cases, since in his spring-arrival series 59 per cent. (84 cases out of 142) developed in the two months following October, as against 30 per cent. occurring during September and October; whereas of the autumn arrivals 57 per cent. (78 out of 134) got it in September and October, and only 31 per cent. (42 cases) in November and December. But

¹ 'Annual Medical Report' to Selangor Government for 1902.

this question of the relation of beri-beri to season will be considered later on.

Evidence from Statistics of Public Institutions.—Better evidence is to be obtained from a consideration of cases occurring in public institutions, where the disease has newly broken out, or where, after it has become endemic, the length of exposure of inmates to the influence (or possibility of infection) before they succumb can be reckoned. Such calculations can be made in two ways. For instance, the total number of individuals exposed for different intervals being known, and taken as separate groups, the extent to which the members of each such group are attacked will afford an index of the relative liability to incur the malady at each successive term. The group of which the largest proportion succumbs will obviously be that which has most nearly been exposed for the period requisite for the average individual to get the disorder. In other words, the interval of possible exposure, during or after which the greatest proportion of subjects is attacked, will mark the average latent period for the average individual. Another and simpler method would be to ascertain in each of the cases occurring in an epidemic what the whole period of *possible* exposure to the influence actually was. Obviously, in the case of a disease like beri-beri—the latent period of which, if in some it has seemed to be three days, in others may be 300—when it is known to be prevalent without as well as within the institution where observations are to be taken, this mode of determination could not be applied to persons admitted during the course of the epidemic; for it would be impossible to affirm of anyone who incurred the complaint under such conditions that any part up to all but the whole of his period of incubation was not undergone outside. But an unexceptionable determination might be made in prisons or asylums when the beginning of the invasion was known by observing the dates at which inmates were attacked who had been in continuous residence in them before the outbreak for such a period as might cover the maximum of necessary exposure. Unfortunately, this analysis has not been undertaken by observers placed in a position to take advantage of such opportunities, and we have at present, therefore, little reliable information on the subject.

In the criminal prisons of the Straits Settlements and the adjacent Native Malay States under British protection committals to the gaols are divided, for disciplinary purposes, into: (1) Short-sentenced prisoners, whose sentence does not exceed six months; (2) long-sentenced prisoners, whose sentence is anything

68 THE CAUSE AND PREVENTION OF BERI-BERI

over that term. The latter are further classified as of (1) *lower grade*, during the first six months of their sentence; (2) *middle grade*, after that term, and until three-quarters of their whole sentence has expired (which may be many years), when they reach (3) the *upper grade*.

A distinction is also made in the returns of *revenue grade* prisoners, who are persons committed in default of payment of a fine, upon discharging which they may be released. These are not classified according to length of sentence, and so may be in for either short or long periods, but, in point of fact, they nearly always serve short terms.

Apart from punishment rations, two scales of diet are in force in all the prisons, which, however, have been varied from time to time. Prisoners whose sentences are under three months have only one—the 'penal diet.' Prisoners committed for longer periods have for the first six months penal diet three days a week, alternated with 'ordinary' diet. After incarceration for six months the ordinary diet is given to all prisoners daily.

The principal distinction in these diets is that no meat is allowed in the penal diet. The physiological requirement of nitrogen is made up with beans, etc.

In the Straits Settlements the amount of rice uncooked in the two diets has been about the same—19 ounces in the ordinary, 20 ounces in the penal diet.

In the Federated Malay States—*e.g.*, in the Selangor prisons—considerable changes in the rations have been made from time to time. The penal diet has never had less than 20 ounces, or more than 22 ounces, of rice daily. But the ordinary diet, which contained only 14 ounces of rice in 1893, was made 19 ounces in 1895, and 21 ounces in 1900.

So far as diet is concerned, the conditions may be taken as alike in the various prisons, while so far as grades of prisoners are concerned, the conditions favour, if anything, the longer-sentenced prisoners. Since all the other factors conceivably capable of determining disease-incidence—air, soil, clothing, exercise, matters of personal toilet and hygiene—are in such institutions the same for all, and (since every prison is rigorously inspected daily) even may be regarded, from the point of view of preventing diseases by sanitation, as optimum conditions, they may be neglected in the consideration of the factors affecting the latent period. In the determination of the latter, therefore, in gaols, the effect of the term of sentence served under the influence of the unknown exciting cause of the disease alone requires to be

considered. Is there any relation observed in gaols between the length of term served and the extent of incidence of beri-beri? It will be seen that the liability to beri-beri depends almost entirely upon the length of sentence reaching a certain term.

Singapore Prison.—In the Singapore Gaol beri-beri seems to have been prevalent almost since its earliest occupation. Rowell, in 1880, gives a table showing that the general mortality through this cause from 1869 onwards had never been less than 2.4 per cent.¹

In 1875, 1876, and again in 1878, the disease assumed epidemic proportions, in the former year causing a mortality of 12.4 per cent., and 8 per cent. in 1876; and in 1878 a rate of 6.7 per cent., which rose to 12.8 per cent. in 1879. Rowell noted that, 'the disease, on its severe outbreak in 1878, in the criminal prison *confined itself to those persons whose sentences were greater than six months*; but during the epidemic of the last four months (1879) it has attacked all grades . . . alike.' Only long sentences are *graded*.

The following table shows the exact number of native long- and short-sentenced prisoners admitted during the years 1878, 1879, and 1880, and the relative incidence of the disease on them:

	Number Incarcerated.	Daily Average Strength.	Cases of Beri-beri.	Case-incidence of Beri-beri per 1,000 Admissions.
1878				
Long sentences	676	469	125	185
Short sentences	2,797	385	17	7
1879				
Long sentences	676	431	271	401
Short sentences	2,099	323	45	21
1880				
Long sentences	515	398	444	862
Short sentences	2,332	294	144	62
1882				
Long sentences	544 }	?	{ 76	139
Short sentences	3,134 }			
			{ 24	7

The case-incidence per 1,000 of daily average strength in either class was:

	Long Sentences.		Short Sentences.	
1878	-	266	-	41
1879	-	628	-	119
1880	-	1,116 ²	-	489

¹ These and further references to Singapore Gaol Statistics appear in the Blue-books of the Straits Settlements for the years concerned.

² Second attacks included.

70 THE CAUSE AND PREVENTION OF BERI-BERI

In 1878 beri-beri was therefore twenty-six times, in 1879 twenty times, more prevalent among the long than the short sentences; or, taking together all prisoners passing through in the two years, we have 5,488 individuals, of whom 26 per cent. only passed more than six months in gaol; and 458 cases of beri-beri, but of these 67 per cent. happened after six months' confinement; from which the inference clearly is that it was at some time after six months that the great majority of the prisoners were most liable to get the disease.

It may be suggested that the greater relative incidence of beri-beri on long-sentenced prisoners is explained sufficiently by the fact that they undergo more numerous chances of 'infection,' every day of confinement, it might be supposed, increasing this chance. But while it may be doubted whether, in a 'focus of infection,' those who have been exposed for, say, thirty days, and have failed to become affected, are any more likely (as a matter of mere chance) to become so in any succeeding thirty days, there is one test which completely disposes of this hypothesis. On the theory alluded to, the proportion of prisoners attacked should have steadily increased *pari passu* with length of sentence, not only up to six or nine months or a year, but beyond it, for as long as the terms might be for which any were incarcerated.

But the facts were otherwise. After a certain period of exposure—i.e., while in the upper grade—the long-sentenced prisoners actually become less liable than even the short-sentenced prisoners to beri-beri.

Of the long sentences those in the middle grade suffered most, both absolutely and proportionately.

The following table shows the *daily average number* in each grade, and the incidence of beri-beri on them :

Year.	Long Sentences.			Short Sentences.		Females.	Total.
	u. g. ¹	m. g.	l. g.	r. g.	s. s.		
1878 :							
Number of prisoners	56	251·5	142·5	65	289	3	807
Cases of beri-beri	3	92	30	2	15	—	142
Incidence per cent.	5·3	36·5	21·0	3·0	5·2	—	17·5
1879 :							
Number of prisoners	49·5	238·5	135	44·5	264	5	736
Cases of beri-beri	14	189	68	5	40	—	316
Incidence per cent.	28·2	79·2	50·4	11·2	15·1	—	42·9

¹ Upper, middle, lower grade; revenue grade and short sentences.

After the lower grade is passed, the diet of all long-sentenced prisoners remains the same, whether in middle or upper grade. No factor of any kind, other than the term of sentence which they had reached, could be discovered to explain the peculiar incidence on middle-grade prisoners in this epidemic.

The fact enables a somewhat closer estimation of the latent period to be made.

Most of those in the upper grade had already served a year. It may be said, therefore, that in this gaol during this epidemic the average latent period in most of the cases lay between six months and a year, with an average of nine months.

There were 588 cases in this prison in 1880, and 229 in 1881, after which the disease gradually died down, and no cases appeared in 1885.

After freedom from the disease for twelve years (1885-1897), beri-beri reappeared (in the autumn of the latter year), and has prevailed more or less severely ever since. The following table gives the number of committals to the prison, and the number of cases of beri-beri occurring among prisoners incarcerated for different terms during this second epidemic :

Committals.						Cases of Beri-beri occurring in					
Year.	Over 5 years.	1 to 5 years.	3 months to 1 year.	Under 3 months.	Total.	Long Sentences.			Short Sentences.		Total.
						u. g.	m. g.	l. g.	f. g.	s. s.	
1896	34	173	350	2,540	3,097	—	—	—	—	—	—
1897	46	171	309	3,367	3,893	—	—	1 ¹	—	2 ²	3
1898	104	176	271	3,532	4,083	7	94	8 ³	3 ⁴	12 ⁵	124 ⁶
1899	92	205	510	2,749	3,556	6	123	6	6	23	164
1900	35	200	251	3,276	3,762	9	105	40	8	52	214 ⁷
1901	135	184	244	3,269	3,832	13	55	40	7	102	217

¹ The first case, a Chinese, on November 20, thirteen weeks after admission.

² The second case, a Tamil, eighteen days ; and the third case, a Malay, six days after admission.

³ One on the twenty-fourth day, one on the thirty-fourth day, two each within five, six, and seven months respectively after admission.

⁴ Two admitted with beri-beri.

⁵ Five at one, two at two, two at three, one at four, and one nearly six months after admission ; one admitted with beri-beri.

⁶ The first four cases were : one, a Chinese, admitted with it ; three Malays, relapsing from earlier attacks incurred outside the gaol—one eight days after admission, one four and a half months, one fourteen months after admission.

⁷ In 1900, four cases were admitted with the disease.

The terms of sentence according to which the committals and cases are grouped not being the same in the Blue-Books and Medical Reports from which these figures are compiled, exact determination of the relative incidence of the disease on prisoners according to all the different lengths of period of incarceration undergone by them before its appearance is not possible. But it is easily seen that in the great majority of cases the disorder attacked principally those who had been exposed to the possible incidence of the cause for periods of six months or more. By placing all those sentenced to more than three months in one group, and assigning to it *only* the cases of beri-beri recorded as occurring in prisoners of the upper and middle grades, and by placing those sentenced to less than three months in a second group, to which all the remaining cases of beri-beri, whether recorded as happening among short-sentenced or low- or revenue-grade prisoners are assigned, we have two groups, one of which shows the *maximum* possible incidence on short-sentenced, the other the *minimum* possible incidence on long-sentenced prisoners.

The following result is obtained :

Incidence of Beri-beri upon Prisoners in the Singapore Prison.

Year.	Short-sentenced Prisoners.	Long-sentenced Prisoners.
	Maximum Number of Cases per 1,000.	Minimum Number of Cases per 1,000.
1898	6	183
1899	12	159
1900	30	225
1901	45	120

The incidence of beri-beri on prisoners serving over six months compared with that on prisoners serving less than six months was, therefore, in this gaol, in 1898, thirty times ; in 1899, thirteen times ; in 1900, seven times, and in 1901, about three times as great.

Again, a comparison of the relative incidence upon prisoners serving for longer and shorter periods more free of error is afforded by calculation of the proportion of cases furnished by each grade of long-sentenced prisoners.

The individuals of these grades being all subject to exactly similar conditions in the prison, from the beginning to the end

of their sentence, the factor of time served can alone be held responsible for large differences, if any, noticed in the disease-incidence.

The following table exhibits the very remarkable differences observed :

Proportion of Long-sentenced Prisoners attacked by Beri-beri in Singapore Prison during the Years 1898 to 1901.

Year.	First Six Months of Sentence (Lower Grade).	Second Six Months of Sentence (Middle and Upper Grades).	After One Year.
1898	7'3 per cent.	86 per cent.	6'4 per cent.
1899	4'4 "	91 "	4'4 "
1900	26'0 "	68 "	5'8 "
1901	37'0 "	51 "	12'0 "

It is clear that the liability to beri-beri of those who had been in gaol for over six months was enormously greater than that of prisoners incarcerated for shorter periods. That this was not due to prolonged incarceration in itself, acting as a factor to increase in some way the predisposition of individuals to acquire the disease, is demonstrated by the fact that so few of the upper-grade prisoners suffered, although they had been in longest of all. The conditions affecting all the prisoners were searchingly investigated by Leask, the medical officer in charge of the gaol during this epidemic, and are fully discussed by him. Nothing, however, was apparent in the occupation, food, clothing, or location in gaol which could be held capable of determining the peculiar incidence of the disease, especially upon middle-grade prisoners. *Their lodging in the prison was altered*, and the cells occupied by them given to others. But while the new occupants remained free where the middle grades had previously been severely visited with beri-beri, *the latter continued to get it in their new location*, the former occupants of which had been free. In short, there was nothing to account for the increased incidence of beri-beri upon the middle-grade prisoners in this prison, except it were what is at once the simplest and most obvious explanation—namely, that it was at or about the period which they must have served in gaol to attain the middle grade (over six months) that the necessary length of exposure to whatever caused the disease was accomplished, and so the symptoms appeared. In other words, the latent period of beri-beri, as it

74 THE CAUSE AND PREVENTION OF BERI-BERI

appeared in the Singapore Prison during the early years of this epidemic, was in most cases not less than six, and in few more than twelve, months.

A closer estimate of the latent period may be made from a consideration of the full report of this outbreak by Leask, who studied it carefully in 1898.

It appeared that, of 124 cases occurring in that year,

There were admitted with the disorder -	-	-	-	-	Cases. 4
There occurred within 1 month after admission ¹	-	-	-	-	5
" " 2 months "	-	-	-	-	3
" " 3 " "	-	-	-	-	2
" " 4 " "	-	-	-	-	1
" " 5 " "	-	-	-	-	1
" " 6 " "	-	-	-	-	3
" " 7 " "	-	-	-	-	3
There occurred after 7 months, but under 12 months, after admission					47
" in second and later years of detention (third to thirteenth), or, say, within not less than 18 months after admission ²					55
Total					124

As the first case which indubitably had its origin within the prison walls in 1897 was that of a Chinese, who showed definite beri-beri on November 30 in that year, thirteen weeks after admission, it is clear that the commencement of the disease in the gaol cannot be placed back at an earlier date than about the spring of 1897. In this case the incubation in the Chinese may have been only thirteen weeks, a very short interval, or it may have been about the average (as we shall see) of eight months, of which half had already been undergone without the gaol.

Analysis of the figures above given shows that, including those cases diseased on admission :

Fifteen per cent. only occurred within the first seven months of possible exposure to the cause in prison, and these were scattered irregularly over the whole interval, the fresh cases in each successive month being, however, always under 2 per cent. of the total.

Thirty-eight per cent. occurred seven to twelve months after admission to prison.

¹ These five were scattered all over the year, and, as Leask says, probably had the disease in them, but not fully developed, upon admission.

² Leask details the number of years' detention in prison of the last twenty-five cases, because he is tracing the possible effect of 'prolonged residence in gaol' (before exposure to its cause) as predisposing to the disease.

Fifty-five per cent. occurred after more than one year—on an average eighteen months after admission to prison.

If from among the cases attributable to the prison be excluded, as Leask thinks (and I agree) they should be, all those occurring within the first month of admission, the proportions would be :

2 to 7 months' exposure in prison 11·4 per cent. of all cases ;					
7 to 12	"	"	40·8	"	"
Not less than 18	"	"	47·8	"	"

or about one-tenth of all cases had a prison exposure of less than six months ; in nine-tenths the exposure was over seven months. In more than one-half of the latter it may have been over eighteen months. So far as these observations go, therefore, the latent period of beri-beri, as it appeared during that season and at that place, may have lain in different cases between extremes of one (a minimum) and twenty-four months (a maximum period), with an average of about one year.

The question naturally occurs here, If in so large a proportion as 98 per cent. of the cases six, seven, or more months were requisite to produce beri-beri, why did any of them occur in less ?

Without invoking a special susceptibility of the individuals, the answer is probably this : Some of the cases are unsuspected relapses, but the rest represent a normal proportion of what may be called 'larval' cases.

There is a certain proportion, it may be assumed, of persons in the population of a country in which beri-beri is endemic who while not having, are yet about to have it. These may be called 'larval' cases. The individuals represented by the term may be considered to be in a sub-beri-beric condition, one which itself causes no illness, is perhaps betrayed by no signs on clinical investigation, but which, if the influences which produce it continue to act, will ultimately become manifested as beri-beri. On the other hand, like any other morbid process, it may cease altogether, never come to the critical point which is revealed by symptoms, if the active cause of it ceases to be applied. The number of persons in such sub-beri-beric state will naturally vary in a community in the same ratio as those with obvious beri-beri do.

In Singapore Island, during the year 1898, 609 cases of beri-beri were treated in Government hospitals, a proportion of

58 per 10,000 of the adult Chinese population, who almost exclusively furnish the cases. The case-mortality was exactly half this ratio. It is impossible to find out exactly how many more cases there may have been who did not find their way into any hospital. But if the proportion of deaths from beri-beri to all deaths out of hospital was the same as in hospital—namely, 21 per cent.—then the death-rate from this cause alone would be about 8 per mille annually; and, since the case-mortality for similar cases cannot very well be taken as greater outside hospital than in, and may very well have been much less, the number of persons affected by beri-beri, to an extent equally with those admitted to the hospitals, may be taken as between 16 and 32 per 1,000 annually, 1 or 2 per 1,000 per mensem. But these cases are not *all* the persons affected. The ratio represents not all those who have been *any* length of time under the influence causing beri-beri, but merely those who have experienced it for the *full period* necessary for it to culminate in obvious disease. That period being taken to be, on an average, twelve months, if the monthly quotient of fully-developed cases is 1 or 2 per 1,000, there must be for each such an one some ten or twenty who are at all periods of development of the malady, short of the production of definite signs, in whom, if they continue to be exposed to its poison, the disease will become manifest.

Were any 1,000 persons to be taken at random from among the population (of Singapore in 1898), and be incarcerated in a place (the prison) where there was beri-beri for a sufficient period for the observation, it would be found, on this showing, that a certain proportion (1 to 4 per 1,000) had declared beri-beri on admission, and that about the same proportion would succumb month by month, as the duration of exposure within the prison, added to that already undergone outside, completed the necessary period of latency in those who, on admission, were at early stages—the larval stages—of the disease. When the average lower limit of incubation (say six months) was reached, many more of those who on admission were free from all signs of it, would begin to show the disorder, and the epidemic would ‘spread’ suddenly about that epoch at some such rapid rate as is indicated in the figures quoted from Dr. Leask.

How closely actual facts fit in with what may have appeared rather loose calculations the following figures show :

The total new admissions to the Singapore Criminal Prison for 1898 were 4,083 (all nationalities).

Of the incarcerations, on admission 4, or 9 in 10,000, had beri-beri.

Of 3,532 persons sentenced to terms of 3 months and under :

In the first month 5, or 14 in 10,000, had beri-beri.

In the second and third 2 months 10, or 37 in 10,000, had beri-beri.

Of 550 persons sentenced to terms of 3 months, 9 months, or 1 year, 110, or 2,000 in 10,000, had beri-beri.

In the last group 102 of the 110 cases had been in for over seven months, so that only eight could have happened in the third to sixth months.

It is remarkable that there was a steady increase in the proportion of short-sentenced prisoners who got beri-beri in this prison year by year, not only relatively to the long sentences, but absolutely. The same process is illustrated in the annually increased proportion of long sentences who acquired it before reaching middle grade—*i.e.*, while in the lower grade, or first six months of sentence.

What the cause of this increased earlier liability to succumb may have been cannot, in the absence of precise information on all heads, be certainly told, but there are several factors which could have had influence to bring about such a condition. One of these is the large number of recidivists among the admissions to prison. Many of the convicts are petty thieves and vagrants, repeatedly convicted to short terms of imprisonment. Constantly in and out of gaol, the several brief terms served by these men, while classed as short sentences, would sum up in effect until equal to the periods served by long sentences.

Another factor which would cause an increased ratio of cases of beri-beri among short sentences would be a change in the proportion of this group incurring sentences nearer to or fully six months. It is likely that prevalence of some particular class of offences should constrain magistrates to sentence offenders of the class at that time to terms longer than had been usual before. Such an increase in sentences seems actually to have happened in 1899, when there was an unusual diminution in the number of sentences under three months, with a corresponding increase in those of from three months to one year and over (*cf.* table).

A more effective influence would be an alteration of the diet, involving a larger proportion of rice and a smaller allowance of proteid foods, as compared with other years. What great effect

78 THE CAUSE AND PREVENTION OF BERI-BERI

even slight alterations in the quantity of rice eaten (when it is toxic) may have will be seen later. I believe that such changes in the diets were actually made.

It will be suggested, naturally, that the increased rate of incidence upon short-sentenced prisoners is more simply explained by increased multiplication and spread of a hypothetical infective agent. But this is negatived by the fact that the incidence upon the long sentences at the same time was not increased, as in such a case it must have been, but diminished.

An increased virulence in the excitant, so that the same effects should be produced by a given dose, but in a shorter time, would also satisfy the conditions. The latent period of the disorder being thereby diminished, a larger number of short-sentence prisoners would fall within the term necessary for producing symptoms.

That the virulence of a living contagion should so increase after years of prevalence is contrary to what commonly characterizes the agents of infectious diseases. It, however, consists naturally with the rice theory that such changes in the latent period—otherwise inexplicable—should occur, since samples of grain may be brought suddenly into consumption more poisonous than those before in use.

Selangor Prison.—In an epidemy which prevailed (as a part of a larger one going on outside) in the Selangor Prison from 1895 onwards, there was similar preponderance in the incidence of beri-beri on long-sentence prisoners.

In 1896, the total committals being 2,560, the number of cases of beri-beri treated was 499. Of these, it appears that 80 (16 per cent.) were admitted with the disorder, 155 (40 per cent.) were relapses from first attacks, or (since they were all classed as separate admissions—readmissions—to hospitals), more properly speaking, recurrences, and the remainder, 264 cases, originated in the gaol. The number of short-sentence committals was 2,459, but only 74 of all the cases of beri-beri were furnished by short-sentence prisoners, upon whom the absolute incidence was thus trifling—under 3 per cent.—whereas the incidence upon the long-sentence prisoners was so severe that all, or nearly all, of them were attacked, many of them twice or oftener. For there were only 99 individuals, or 3·8 per cent. of all committals, in this class added in this year to those remaining from the previous year (under 150). But among them 103 individuals were attacked for the first time, and there were 155 relapses.

In 1897 the committals were 2,670, the number of long sentences 126, or 4·7 per cent. The admissions for beri-beri to the

prison infirmary were 276, of which 200 are stated to have been 'relapses.' The number of persons entering the prison with the disease is not given in the reports to which I have had access, but may be assumed to be the same as in the previous year—viz., 16 per cent. The fresh cases arising in the gaol would thus be about 64. McClosky,¹ the medical officer in charge, reported that only 52 out of all the cases were furnished by short-sentence prisoners, of whom 2,544 were committed during the year, so that the absolute incidence upon them in that year was under 2 per cent., even including among them the cases which were affected on admission.

Were such a proportion of these latter deducted as would accord with the greater preponderance of the short sentences to all admissions (of which they form 95 per cent.), then the incidence upon short sentences, the extent to which they acquired the disease in prison, would be 1.7 per cent.

The incidence on the long-sentenced men was more than cent. per cent., 245 cases being furnished by them, of which, however, 200 are stated to have been recurrences, or relapses.

Travers, in a paper presented to the Selangor Government, analyzes 386 cases occurring in this gaol during an interval covering a part of each of these years—namely, from October 1, 1895, to June 30, 1896. The same period and exactly the same cases are dealt with in a table, with remarks, compiled by McClosky, and printed by H. Wright.² The number of *individuals* furnishing the 386 cases considered was, Travers states, 279. Of these individuals 122 were long-sentenced, and 157 were short-sentenced men. On this showing only, he concludes that short-sentenced men were equally, if not more, liable to get the disease than long-sentenced men.

But McClosky's table shows that 86 of all the individuals affected had the disease *on committal to prison*. Since only 5 per cent., or less, of all committals to the prison are long sentences, it follows that some 95 per cent. of the prisoners affected on admission must have been short sentences. Some 80 or more of the cases admitted with the disorder must, therefore, be deducted from the tale of 157 short-sentence individuals whom Travers gives as having the disease to obtain the proportion of them who acquired it *in gaol*.

There next have to be imported into the calculation the figures (which Travers omits) showing the total numbers of short- and

¹ *Selangor Government Gazette*, 1898.

² 'Study,' p. 39.

long-sentence prisoners respectively committed to the gaol during the period reviewed, from among whom the actual patients were drawn, in order to find the *relative* proportion of cases which each class furnished. Now, there were committed some 120, and passed through the gaol altogether *not more than 200 long-sentence prisoners*, not *less than 2,500 short-sentence prisoners*, during the term of ten months.

The result calculated from all the figures is that the absolute incidence upon the short-sentence prisoners during the interval reviewed could not have been more than 3 per cent., while upon the long-sentence prisoners it could not have been less than 49 per cent., and may have been as much as 80 per cent., without counting relapses.

Of short-sentence prisoners attacked, 17 per cent. had relapses, of long-sentences no less than 77 per cent.

Of long-sentences attacked, 34 per cent. became affected while in the lower, 60 per cent. in the middle, only 6 per cent. while in the upper, grade. Many of those attacked in the lower grade, it is to be remembered, may have approached near to the expiry of their term, and so may many of the short-sentence prisoners attacked. This will make the true proportion of those attacked at or about the sixth month of incarceration correspondingly larger. But it would seem that in this prison at this season the latent period exhibited, or probable, in the majority of cases, although according closely with that observed elsewhere, was perhaps somewhat shorter than six months.

H. Wright¹ (whose observations on 'sinkhehs' have already been cited) and Travers² have each attempted to show that the usual latent period is short, from analyses of the dates after admission at which patients, prisoners at Pudooh Gaol, individually acquired the disease.

Wright analyzes 90 such cases occurring among 1,293 prisoners passing through the gaol between May 3, 1901, and April 1, 1902, all of whom had first been examined and passed as healthy by himself. Although the method used to determine the point by these observers is incorrect, while a much better one was at their disposal, I give the tables, both for what they are worth and also because, when supplemented by the proper data, they afford valuable confirmation of the views already unfolded as to the peculiar increase of incidence with lengthened incarceration.

¹ *Loc. cit.*, p. 54.

² 'Annual Medical Report' for 1902, *Selangor Government Gazette*.

Travers deals with 256 fresh cases of beri-beri originating in Pudoh Gaol between January 1 and October 31, 1902.

Table showing Period of Exposure (or possible Duration of Latent Period) of Prisoners who acquired Beri-beri in Pudoh Gaol.

H. WRIGHT.		TRAVERS.	
Days of Incarceration.	Number of Cases.	Days of Incarceration.	Number of Cases.
1 to 7 days	3	Under 14 days	4
7 " 14 "	10	14 days to 1 month	7
14 " 21 "	6	1 month to 2 months	45
21 " 30 "	6	2 months to 3 "	67
30 " 45 "	9	3 " 4 "	40
45 days to 2 months	10	4 " 5 "	27
2 months to 3 months	11	5 " 6 "	25
3 " 4 "	12	6 " 7 "	8
4 " 5 "	8	7 " 12 "	13
5 " 6 "	6	Over 12 months	20
6 " 7 "	2		
7 " 8 "	1		
8 " 9 "	1		
9 " 12 "	2		
12 " 18 "	3		
Total	90	Total	256

Wright concludes from his series that the incubation period 'may' in some instances 'be as short as ten days.'

Travers, observing that 'of 252 prisoners contracting beri-beri, 63.5 per cent. were attacked before they had been four months in gaol . . . 86.9 per cent. before seven months, and only 13 per cent. after seven months,' infers that 'the conclusions drawn by Dr. Braddon as to the incubation stage of beri-beri at the Pudoh Gaol are obviously without basis.' It will now be seen why analyses made after the method of these two observers are futile.

What is wanted to be known—the question which the material available at Pudoh might, otherwise handled, have correctly answered—is not, How many, out of a given number of beri-beri patients, acquired their disease at given intervals after admission to prison, but, Of the total prisoners incarcerated for each such interval, how many got beri-beri?

For (1) seeing that the disease prevailed equally without as within the gaol, and that many cases even entered the gaol with

beri-beri, it could not be affirmed of any single case in either series that the disease was not wholly or in part acquired by the patient outside the gaol, whether he appeared healthy when first inspected after committal or not. If the disease be an infection it is impossible but that some of the cases should have been infected outside, their symptoms only becoming manifest after entering prison, whatever the length of the incubation period might be, while if the disease be a chronic intoxication this would have been yet more the case.

(2) Had the total number of prisoners incarcerated for each of the successive terms in the tables been stated, the very different picture would have been presented of a rate of incidence of beri-beri steadily increasing *pari passu* with the length of the period of confinement.

It happens that the same cases dealt with by Travers here appear again in the table (already referred to) published by him later,¹ which purports to show that the incidence of beri-beri was greater on short- than on long-sentenced prisoners.

In this last table all the cases occurring in the prison during 1902 are analyzed to show the proportion occurring in long- and short-sentenced prisoners. The table is introduced into his report by Travers as follows :

'The following analysis of cases² of beri-beri in the gaol, showing the numbers of new and relapsed cases, long- and short-sentenced prisoners, with rainfall and division of monsoons, will, I think, be of interest.'

It appears that 890 cases altogether were treated in the prison infirmary, of which 415 were relapses, 475 'new' cases.

Of the 475 'new' cases, all but 25—i.e., 450—are given in this table as admitted to the infirmary between January 1 and October 31, 1902. But of these the observation cited above (on p. 80) shows that only 256 *originated* in the gaol.³

One hundred and ninety-four therefore—i.e., 450 less 256—had the disease *on entering the gaol*.

In Travers' table the distinction between cases so coming in with beri-beri and those acquiring it after admission is not made. They all appear together as 'new' cases. Nor is any reference

¹ 'Annual Report of Medical Department, Selangor, in 1902,' p. 3.

² See next page.

³ In his 'Further Observations on the Rice Theory,' etc., Travers states : 'Between January 1 and October 31 of 1902, 256 *fresh cases of beri-beri originating in the Pudooh Gaol* were admitted to the infirmary.'

made to the fact that the short-sentence prisoners committed outnumbered the long-sentences by 20 to 1.

By this arrangement it is made to appear that the incidence of beri-beri was greater on short-sentence than on long-sentence prisoners, since, out of the 450 'new' cases occurring between January and October, 186 only are recorded as belonging to the latter, against 264 in the former class. But taking the proportion of long-sentenced prisoners to all committals in this gaol—namely, 5 per cent.—there would be certainly not more than 150

Beri-beri at Kuala Lumpur (Pudoh) Gaol, 1902.

MONTH.	NEW CASES.		RELAPSED CASES.		RAINFALL.
	Long-sentenced Prisoners.	Short-sentenced Prisoners.	Long-sentenced Prisoners.	Short-sentenced Prisoners.	
January	20	26	18	4	Inches. 3'77
February	8	29	4	3	10'24
March	12	34	10	7	9'65
April... ..	22	36	13	9	11'36
May	25	42	22	2	11'28
June	34	20	26	3	4'55
July	21	18	25	3	2'98
August	12	15	42	6	4'91
September	14	23	76	18	8'60
October	18	21	48	11	20'89
Total for ten months }	186	264			
November	5	4	20	6	18'07
December	3	13	31	8	10'10
	194	281	335	80	116'40
	475		415		
Total for the year }	890				

long-sentenced prisoners committed, or more than 200 passing through the prison during the ten months of observation, who could be subjects of beri-beri.

Since the long sentences actually furnished, on Travers' showing, 186 individual cases, the incidence on them must have been, as in other years, nearly total—93 per cent.

The average number of short-sentenced prisoners passing through in the same time was more than 2,500, producing, according to Travers' showing, 264 cases, or 10½ per cent.

But the real disparity was even greater than this, for the

84 THE CAUSE AND PREVENTION OF BERI-BERI

number of new cases in all classes *originating* in the gaol was, as has been shown, not 450, but 256—194 were admitted to prison with beri-beri. To obtain the proportion in which these 256 cases were distributed between the two groups of long and short sentences, we have first to ascertain the proportions of the same classes in the 194 prisoners admitted to gaol with the disorder; these then have to be deducted from the corresponding totals of the 'new' cases among long and short sentences as given by Travers. The proportion of long sentences to all committals annually in the gaol was 5 per cent. Only 5 per cent. of the cases admitted with the disorder, therefore, can be assigned to the long-sentence class; the remainder go with the short-sentence class. Of the 194 patients admitted to gaol with beri-beri, then, 10 actual cases belong to the long-sentence class, 184 to the short-sentence class. Deducting these from the total 'new' cases given by Travers as treated in either class during the period of his table, we have the following results:

New Cases of Beri-beri Treated in the Pudooh Prison between
January 1, 1902, and October 31, 1902.

	Long Sentences.	Short Sentences.	Total.
Cases admitted to prison with beri-beri	10	184	194
Cases which acquired beri-beri after admission to prison	176	80	256
Total 'new' cases as in Travers' table ...	186	264	450

Taking the same number of committals as before, the proportion of individual long- and short-sentence prisoners who acquired beri-beri in this prison in 1902 appears as 90 per cent. and 3·8 per cent. respectively.¹

An interesting statement as to the frequency of *relapses* is given by Travers in the same table.

Of 475 individuals admitted to hospital with beri-beri, 415 subsequently had relapses.

¹ Two hundred being total long sentences passing through during the whole year, the number for the ten months only would be less—say 180—among whom originated 176 new cases = 90 per cent. Of the short sentences, 2,082 are estimated to have passed through in the ten months, producing *eighty* new cases acquired in gaol, or 3·8 per cent.

The long-sentence cases admitted during the year—194 in number—furnished 335 relapses. The short sentences—281 in number—furnished 80 relapses. Convicts of the latter class, of course, spend too short a time in prison to incur relapse.

The enormous increase in the number of cases in this gaol in latter years seems to call for remark. Not only are the relapses more numerous among long sentences—nearly twice as many as the original cases—and among short sentences also, but the proportion of those entering the gaol with the disorder shows an increase greater than any observed in the hospitals outside.

It seems likely that here, again, some explanation of the disproportionate increase in gaol cases may be found in the steadily-increasing number of recidivists. Half the committals to the gaol in any one year seem to have been already admitted once before; half of these, again, had been thrice committed. Many of the cases apparently incurred in gaol for the first time may thus very well be recurrences after primary attacks incurred there during a previous sentence. I quote the following statistics as to recidivists from official records:

Proportion of Recidivists to Total Convictions in Pudooh Gaol,
in Percentages of Total Convictions.

	1898.	1899.	1900.	1901.	1902.
One previous conviction only ...	48	50	50	52	54
Two previous convictions ...	23	20	18	17	20
Three previous convictions...	27	26	31	29	23

Inference as to Average Latency Period.—To sum up, the figures cited in connection with these two gaols prove that in them the rate of incidence of beri-beri upon those incarcerated increased steadily with the length of the term served up to six months or thereabouts; that it diminished after that term; and that the altering incidence was not due to the effect of imprisonment itself operating as a factor *per se* to increase predisposition to the disease.

The inference would appear to be sound, and (in the absence of any other demonstrated or probable factor to determine the event) must be adopted, that the increased relative incidence at or about six months must be due to the fact that that period is, for the great majority of persons, about the term required for the influence which produces the disorder to manifest its effects.

86 THE CAUSE AND PREVENTION OF BERI-BERI

In other words, the latent period of beri-beri, as it appeared during those seasons and at those gaols was, in the greater number of cases, about six months, in many perhaps a little less, in few more.

Confirmed by Statistics of other Institutions.—The only other statements I have been able to find bearing on the period of latency as observed in public institutions are :

1. One by Leslie,¹ who, in reviewing the epidemics of beri-beri in the gaols of Burmah, records that at Thayetmyo Prison in 1881 no prisoner was attacked whose term of sentence was under two months.

2. Dr. Conolly Norman's observation that two of the patients attacked at the Richmond Asylum, Dublin, in 1897 (one of them a nurse), had only been in the asylum six weeks before being attacked (*British Medical Journal*, 1897, vol. ii., p. 168).

3. Vorderman² found the interval between admission to gaol and getting beri-beri in it to be on an average 106 days. In eight specified places it ranged between 92 and 120 days.

4. In the Dakar Prison in Senegal, Lasnet, describing one outbreak occurring among prisoners,³ and confined to the aborigines, says: 'Of thirty-two soldiers who had been confined upwards of five months, two-thirds became affected. . . . The affection generally commenced towards the third month of incarceration, with malleolar œdema, constipation, general depression, etc.'

Among the remaining 545 native prisoners confined for short periods only 13 cases occurred—2.2 per cent.

Lasnet attributes to want of exercise the chief influence in determining the incidence, since of the military prisoners who were not compelled to work, but lay idle all day in prison, 16 per cent. were attacked (24 cases among 149 men), whereas among the civilians who were sent out to work less than 5 per cent. got beri-beri (21 cases in 446 men). He also remarks that on compulsory exercise such patients improved, but if the patients remained in prison a relapse ending fatally was sooner or later certain to supervene.

It seems more probable that difference in the proportion of civilians incarcerated for longer periods accounted for the ob-

¹ 'Notes and Statistics of the Administration of Hospitals and Dispensaries in Burmah,' Government Printing Press, Rangoon, 1897.

² 'Onderzoek,' etc., p. 63.

³ *Archiv de Méd. Nav. et Col.*, tom. v., 1897. Reference: *Lancet*, April 3, 1897, vol. i., p. 973.

served difference in incidence of beri-beri upon them, but the facts about this are not given.

5. Stanley¹ states that, in the four Shanghai gaols, where he observed 500 cases during 1898-1901, they occurred almost entirely among those who were sentenced to over one month, 14 per cent. of whom were attacked.

6. Dykes² says that at the Gauhati Gaol, Assam, the shortest periods noted were: one six weeks, one thirteen weeks after admission.

7. Gerrard³ analyses 210 cases, in which the date of appearance of the disease varied from twenty-three days to over 1,000 days after admission to Pudooh Gaol in 1896-1898.

Ucherman,⁴ as the result of independent investigations, carried out in reference to over 100 ships upon which epidemics occurred, has arrived at a similar conclusion as to the incubation period. He considers that there is no indication of one.

These Results incompatible with the Infection Theory, but consistent with the Rice Theory.—The prison epidemics support, therefore, the conclusions gained from other sources, and proving the deduction from the rice theory, that there is no fixed period of incubation of beri-beri. The period for which the poison may act before producing disease is irregular, and may vary within wide limits, which depend on factors not at present certainly known, but the most important among them may be surmised to be the dose of toxic agent absorbed. I shall not dwell here upon the anomaly of a presumed infection, of which the incubation period may be at one time no more than four days, at another perhaps a year, and which in either case results in the same acute event. Those may explain it who believe beri-beri to be due to an infection. The limits within which the latent period may vary, exceeding as they do anything known of diseases certainly ascribable to living germs, oppose such a theory of its causation as strongly as they support the view which is developed in this paper, that the disorder is nothing more than an intoxication, the slowly accumulated effect of small, or the rapid result of large, doses of poison repeatedly consumed.

The process may be, according to the quantity and intensity of the dose of noxious material taken, in some cases one merely of hours, in others of years. But, although there is no regularity

¹ *Loc. cit.*

² *Indian Medical Gazette*, June, 1904.

³ *Beri-beri*, 1904, p. 59.

⁴ *Lancet*, p. 26, 1902.

in its period, there can be little doubt that, under the circumstances in which it is ordinarily incurred, the interval is in the vast majority of cases prolonged—seldom less than three, usually more than six, and often more than eight or nine months.

No experiment, therefore, by which it is proposed to ascertain the part played by any questioned etiological factor in the causation of beri-beri can be relied upon to provide definite evidence in which care is not taken that the individuals concerned are, all of them, kept under continuous observation for a period which shall cover at least the ordinary term of latency of the disorder, and preferably the maximum of a year.

Erroneous Conclusions due to the Failure to Recognise the Possible Extent of the Latency Period.—The importance of recognising the great length of the latent period of beri-beri has been conspicuously illustrated in several observations made in connection with *gaols*.

Thus Rowell,¹ discussing the etiology of the disease in connection with the great epidemic at the Singapore Prison in 1880, sums up his reasons against *food* at that date as follows: 'I cannot look upon the diet in any way as a factor in the causation of the heavy sickness in the gaol. The female prisoners, who received the same diet, thrived on it. The prisoners in the sister settlements (Penang and Malacca) were on the same diet, and it was the same for the civil prisoners sent to the Singapore Gaol for treatment. Yet there was (with a brief exception at Penang) no beri-beri among any of these other prisoners.'

So stated, the conclusion drawn by Rowell seems inevitable.

¹ It is but fair to add that this distinguished medical officer at a later date announced altered views as to the connection of beri-beri with diet in this gaol. In his Report to Government for 1885, he writes: 'Looking back on the past, and having watched carefully the state of the prisoners' health throughout, I feel constrained to admit that a certain share of it [the prevailing unhealthiness prior to 1885] was occasioned by a faulty dietary.' Rowell is even at that date so far from ascribing the real influence to rice, that he says (p. 125): 'In beri-beri epidemics, occurring whether in India, Japan, or elsewhere, a theory has every now and again cropped up that the disease may be owing to unwholesome epiphytic rice, *but the gaol experience does not tend in any way to support it*. Every kind and variety of rice, all of the best quality, had been tried during the continuance of the disease' [for how long a term?], 'but with no difference as to result.' [Yet the disease fluctuated, not merely annually, but monthly.] 'I would be much more disposed to believe, on the other hand, in the truth of a doctrine which would give to a diet too poor in its nitrogenous constituents—such as ours unquestionably was—a large influence in this direction by predisposing the body to the reception of the poison, whatever the peculiar nature of that virus may be (Straits Settlements Blue-book, 1886, C. 30).

If, at the same period, for several years, the inmates of three similar institutions—gaols—all had the same (or similar) food, it is clearly against the probabilities that any disease due to the food should have remained entirely confined to one gaol only of the three, supposing all other circumstances to be equal.

But the circumstances were not equal. The factor of the long latent period entailed, as it happened, a certain difference between the gaol at Singapore and the other two gaols.

It has been shown how, as a result of this factor, the incidence of the disease falls almost exclusively upon long-sentence prisoners. In Singapore in 1878 only $\frac{1}{2}$ per cent., and in 1879 only 2 per cent., of all the short-sentence prisoners were affected. The incidence on the long-sentence men was 18 and 40 per cent. respectively. It follows that the relative liability of a *prison* to beri-beri will depend on the number of long-sentence convicts it contains. Were no long-sentence prisoners kept in a prison there would be no beri-beri. Now, this was the case with the Penang and Malacca prisons.

All their long-sentence prisoners, it appears, were sent to serve their term in Singapore.

In this simple manner the great discrepancy in the liability to beri-beri of the three gaols is at once explained. Although they were all using the same food, the inmates were not fed on it sufficiently long in each institution for the disease to develop.

It may be suggested that still a few cases should have happened among the short sentences in 1880. Probably they did. But 2 per cent. of cases, and those very likely of the mildest, might readily go unobserved. Attention is drawn to such ambulant cases elsewhere (see p. 6).

That the above explanation of the facts disposes of Rowell's objection to the food theory of beri-beri causation seems proved by the following episode, which has been widely quoted by those who see in it some evidence in favour of infection :

Although the Singapore prison had been more or less continuously infested with beri-beri since its opening, that at Penang had always been free. In 1880, in order to give the stricken inmates of the Singapore gaol such benefit as change of location could secure, the long sentences (216) were transferred—180 to Penang, and 36 to Malacca. The long-sentence prisoners, usually sent to Singapore from Penang and Malacca, were all kept at those places. The transfer was made in April. There is no record of any unusual occurrence at Malacca, but *two months after the transfer an epidemic of beri-beri broke out* (for the first

time and last) in *Penang prison*, lasting till July. Sixty-one cases occurred, with two deaths.

Rowell, after careful investigation on the spot, reported that there was no ground for supposing the Penang epidemic to be in any way the result of contagion. As must by now have become evident, the occurrence was merely due to the fact that on this occasion (when the cause of beri-beri was everywhere active) there were for the first time at Penang prisoners in whom the conditions permitted its development during their confinement in that gaol.

Travers' and Wright's Experiments at Pudo.—Two experiments made at the Pudo Gaol in Selangor, by Travers in 1895, and by H. Wright in 1901, have, through the same fallacy, been similarly erroneously interpreted by these authors, as affording evidence against food as a factor in beri-beri causation. More detailed reference to the observations will be made later on. In Travers', the prior observation, two parties of prisoners were kept for eight months at different gaols on the same food; but as it happened that most of the long sentences were kept in one, and the short sentences in the other gaol, beri-beri was only observed in the gaol where the former were confined. Travers, not having taken this factor into his reckoning, esteemed both parties as equally circumstanced, and hence was led into the belief that, the food supplied to both being the same, it could play no part in determining the different incidence of beri-beri.

H. Wright, whose experiment has already been briefly referred to, repeated exactly the same proceeding, only with application to two separate parties confined in one and the same gaol.

There will now be considered in order those deductions from the rice theory which, depending essentially on *rice*, can be derived from no other hypothesis, and the proof of which is the logical proof of the theory.

NOTE.—The singular increase (referred to on pp. 76, 84) in the proportion of short sentences attacked by beri-beri as compared with long sentences has been treated in the text as being a real and not a merely apparent increase. Since the MS. was closed, however, further consideration of all the circumstances, on the spot, has convinced the writer that the disproportion in the relative incidence on the two classes of prisoners in later as compared with earlier years of the epidemics was less real than apparent. The fact was that with increasing familiarity with the disease, and the exercise of minuter care to detect cases, patients became sooner diagnosed, and many slight cases went to swell the record such as in earlier years went unobserved. The effect of this will be to make the comments in the text applicable in former years only to well-marked cases, and to reduce the latent period for the development of the earliest signs.¹

SECTION IV

PROOF OF DEDUCTIONS MADE FROM THEORY OF GRAIN INTOXICATION IN CASE OF RICE

I

AMONG MIXED COMMUNITIES, AND IN PLACES WHERE BERI-BERI IS PREVALENT, RICE-EATERS ONLY ARE ATTACKED; THOSE WHO EAT NO RICE ESCAPE

Direct Relation of Beri-beri to Rice-supply.—Expressed in general terms, the chief conclusion flowing from the rice theory, which can be derived from no other premises, is this—that *beri-beri should show a direct relation to rice-supply*.

It should be present where rice¹ is eaten, it should be absent where rice is not eaten; and variations in its incidence and severity should be concomitant with definable variations in the rice-supply.

That beri-beri attacks rice-eating races, and does not attack people fed on other grains, has as a vague general observation long been accepted. But since the countries in which the disease is endemic are also those in which rice is grown, and is naturally the staple food of the inhabitants, this observation could at best do no more than raise a presumption that rice might be the cause; while the undoubted fact that there are great areas of country where rice is grown, and forms the staple food of the inhabitants, in which beri-beri is not endemic, nor even of frequent occurrence, raised an insuperable obstacle to accepting rice *qua* rice as a cause of beri-beri.

¹ 'Rice, including its derivatives.' For the present, beri-beri is under consideration only so far as it may be caused by a poisonous concomitant in rice itself used directly as food. Beri-beri caused by rice derivatives—articles in which rice is present as an adulteration, or which are extracted from rice (starch and glucose)—or by other cereals attacked with the rice fungus, will be considered later.

But it is required by the present theory (1) that only that rice which is grown in a country where the cause of the disease is indigenous, or which the same poison has subsequently infected, shall be capable of causing the disease, and of causing it, moreover, in any other country to which such rice may be carried; (2) that rice prepared in particular ways should be, even if poisonous as grown, rendered either wholly innocuous or at least less injurious than grain not so prepared.

The exceptions, large as they may be, to the first general conclusion will be seen to be readily explained by these conditions later on. In the meantime it will be advantageous to consider the particular modes of the general proposition, and establish them one by one.

In epidemics of beri-beri, and in places in which it is endemic, rice-eaters only are attacked; those who eat no rice escape.

Striking examples of this fact have long been before the profession. There is no lack of illustrations.

Malcolmsen¹ long ago (1835) noted differences of this kind. Thus he gives statistics to show that in one regiment, the 38th N.I., the Musalmans 'suffered more than the Hindus,' the case-incidence in the former being 12.7 per cent., as against 6.27 per cent. among the latter. He suggests the difference to be due to the more generous diet eaten by the Musalmans. 'The Bengal Sepoy, who shuns animal food,' he also says, 'and who is far more sickly than the Madrasi, yet does not get beri-beri.' He observes that one difference between the two classes was that the Bengali ate sparingly, and of *wheat* only, never rice. This remains true to-day.

Evidence supplied by Ships.—The numerous instances of the crews of ships in which, after a more or less prolonged voyage, the disease has broken out, afford other examples. It is almost invariably the case among such crews that those who are attacked are natives; when not natives, Europeans partaking of native diet, including shop-rice which is bought at the port of clearance for the voyage.² The cheapest (not necessarily a bad sample) in the market is naturally bought, and in the East this means that the variety used is almost always Burmese, Siamese, or Cochinese. Neither, in the case of lascars, natives of India proper, Malays, or Japanese, is any one of these the sort of rice, nor in the case of Europeans even the class of food, to which they have been accustomed.

¹ 'A Practical Essay.'

² The case of beri-beri in European ships and crews will be dealt with later.

But they are precisely the sorts upon which those feed systematically who are wont, as will presently appear, to get beri-beri. That the disease should break out only after, as is usual, some months at sea is to be explained by the long incubation period, the slowness of the development of symptoms from absorbing a poison, the effects of which are accumulative and become only gradually manifested.

The complicated view which, in the absence of evidence to incriminate food, Manson and others have adopted, that these cases of ship beri-beri are miasmatic, produced by some organism which clings to holds and cargo, to the walls of cabins, and especially of reeking forecastles, is, in the face of this simple explanation, unnecessary.

The fact that for centuries the crews of thousands of ships engaged on voyages as long, and pent in cabins even more confined and steamy than those occupied by the hands on well-found ships of the present day, but not fed upon rice, have not been wont to get beri-beri alone confutes the miasmatic theory. Again, the beri-beri ships have been cleaned and really thoroughly disinfected after the appearance of beri-beri in them, everything found new, and fresh crews engaged, and yet beri-beri has persistently remained, or reappeared in them, voyage after voyage, and it is claimed against the probabilities that there must be a permanent contagion. The explanation is much simpler.

The error has been that the provisioning has been always of the same character. The same sort of rice has been supplied, in most instances from the same port, and often enough no doubt from the same contractor. At times this rice has been poisonous, at others not. The following cases are cited in proof :

(i.) Major Anderson,¹ describing the outbreaks of beri-beri in the ships of the Indian Marine Survey, gives convincing evidence as to the connection of the disease in them with *the food*. Every year from 1894 to 1899 cases occurred in one or both of two ships in service, after putting to sea, so that there was ample opportunity for studying the conditions involved. Anderson, in summing up the conclusions come to, says : ' Cases of the disease occurred in all classes of the crews, except the officers, *whose food is obtained from a totally different source* from that of the warrant officers, lascars, and stokers. It affected the healthiest and the weakest, the old and the young. It has broken out in the cold, dry, windy weather of December on the Indus coast, also in the hot, moist, still atmosphere off the mouth of the Bay of Bengal

¹ *Loc. cit.*

in the month of November. The one feature common to all the outbreaks has been that they have occurred within two, and generally within one, month of leaving Bombay, and always while Bombay provisions were being used.' In describing the epidemic on the two ships in 1894, Anderson says: 'Finally, as all other measures failed [and every possible remedy seems to have been tried], on December 14 the remainder of our stock of Bombay rice, dal, flour, etc., were hove overboard . . . a fresh stock obtained from Karachi, and the disease at once ceased.

. . . That the cause of the disease was contained in the food is rendered highly probable by the immediate cessation of cases on changing the food-supply in the ship [the *Investigator*], its continuance in the detached boat parties when using the suspected food, and its stoppage in them and the *Nancowry* [the *Investigator's* consort] when the new supply of provisions was obtained.'

No one who reads his report can fail to agree with Anderson that the cause of beri-beri in this epidemic lay in the food.

Now, the rice was the only part of the food which it is reasonable to suppose could have been at fault, since it was not only the principal article of diet of the crew, and that in use of which they differed from the officers, but it also was the only article among the many consumed common to them and the thousands of persons who in other instances acquire beri-beri.

It will not prejudice this conclusion to add Major Anderson's statement in his article that 'bad feeding, *mouldy rice*, heat, moisture, insanitary conditions, scurvy, and, latterly, alcoholism, all supposed causes of beri-beri, were entirely *absent* in this epidemic.'

'Mouldy rice'—*i.e.*, visibly mouldy rice—as Major Anderson here conceived it, doubtless there was not. But rice there was. And the change in a rice which renders it toxic so as to produce beri-beri is, by the present theory, not necessarily manifest to the naked eye, or even to the microscope, or in any other manner than its results upon the consumer.

(ii.) A record even more convincing than Anderson's is that by Haynes¹ of epidemics of beri-beri occurring in the Cossack (Australian) pearling fleets, where the manner of its incidence upon mixed nationalities, according as rice was or was not eaten by them, leaves hardly any room for doubt that it was through consumption of this article that the disease was incurred.

Haynes gives an account of the epidemics occurring in seven cruises between 1883 and 1887. The crews were principally

¹ *Loc. cit.*

Malays, in number between 70 and 118. Their diet was rice and fish, supplied from a port in the Malay Archipelago. In addition, many Japanese, Australian natives, and South Sea islanders were employed. The staple food of the former was 'damper,' of the latter 'taro'—yams.

On every voyage except the fourth and fifth the Malay crews were severely visited by beri-beri, and there were many deaths. The earliest date of appearance of the disease was the third, but the usual one, or that after which most cases occurred, the seventh month. The work was much the same for all. 'The ventilation at night favoured rather the Malays.

'The numerous . . . Australian natives employed . . . *fed upon damper* . . . were entirely exempt from the disease, . . . the South Sea men, who are bred [and were hence always dieted] on yams, appeared entirely proof against it, as also were the Europeans.'

In the exceptional voyages upon which no cases happened 'a mixed diet was adopted, and care taken to send the men home before the seventh, the fatal month, was out.'

The conclusions offered by Haynes, and which his facts seem amply to warrant, are expressed as follows: 'Nothing has been reported from subsequent voyages to alter the opinion:

'(a) That beri-beri is confined to a very great extent to rice-eating races, and with proper care will not develop in less than seven months.

'(b) That the substitution of a mixed diet of wheat-flour, beans, potatoes, etc., *to the exclusion of rice*, mitigates, if it does not entirely prevent, the disease.'

Here, again, were conditions affording the most favourable opportunity for ascertaining the true causal factor of beri-beri. A number of persons, all engaged in the same occupation, for a length of time which was sufficient for the disease to develop, separate from all chance of stray infection, absolutely cut off from land and hypothetical soil and miasmatic emanations, of mixed nationalities, all healthy to start with, and all subject to external conditions identical in every respect, save only the character of the food consumed. The incidence of the disease was upon those only who ate rice. Of five nationalities, two, consumers of rice, got beri-beri; three, who ate no rice, escaped. Moreover, among the rice-eaters already affected, the disease disappeared when the rice was replaced by other articles or fresh supplies.

Were beri-beri not a disease which there seems to have arisen an obsession on the part of its investigators to regard as mysterious, such instances as these would long ago have been accepted as sufficient indication of its source and nature.

(iii.) The freedom from beri-beri enjoyed by the Australian natives while fed on 'damper' in the epidemics described by Haynes on the pearling ships is rendered the more interesting through an account by Weatherall of an outbreak among natives of the same race in Australia upon an occasion when the individuals concerned were fed upon rice.

Weatherall¹ narrates that sixty aboriginal native prisoners were under his care at Wyndham, Kimberley, in Western Australia. They were housed in an iron shed, ill-ventilated, in a tropical climate. Their diet was one pound of bread, three-quarters of a pound of meat, generally salted, *half a pound of rice*, three pints of sweetened tea, without milk, daily. 'Occasionally broth was served, and the rice was supposed to act as a substitute for fruit and vegetables, which were not procurable. It must be remembered that the natives were accustomed to roam unclothed, wild in the bush, . . . living on kangaroo, iguana, honey, fish, roots, barks, etc.

'Under these conditions a number of them . . . developed beri-beri, the principal feature of which was the extensive oedema and liability to sudden death, apparently from syncope. The white inhabitants of the town were none of them affected.² Beri-beri could not have been introduced from without, for there was no one either entering or leaving the place, a steamer calling but once every two months. It must have had, therefore, a *de novo* origin, and is, so far as I know,' says Weatherall, 'the first time beri-beri has ever been seen in the aboriginal natives of Australia.'

(iv.) In the same paper in which this account is given by Weatherall appears one by Orr of six cases of beri-beri occurring among lascars on a P. and O. vessel running between Italy and Egypt during the cold weather, November and December, 1893. The diet of this crew was 'chiefly curry and *rice*.' Orr, commenting on the possibility of contagion, says that 'none of the men had

¹ *British Medical Journal*, September 28, 1895.

² In a later note Weatherall says the rice used was the same as free natives in the town also used. This fact does not indicate that it was therefore not toxic, only that when freely diluted with other articles of diet—*i.e.*, when eaten in *relatively* less quantity—it was innocuous.

been out of the Mediterranean for six months, so that the only possible source of contagion would be from crews of other ships at Brindisi. No European suffered from the complaint.¹ Orr's case will serve as one example out of many such that annually occur in British ships where lascars are employed.

(v.) One of the most remarkable among these ship epidemics is that narrated by MacLeod, as occurring on a sailing-ship, the *Ancona*.¹

The ship was of steel, and previous to the outbreak had been only in the Clyde, Australia, Portland, Oregon, and New York. She left the last port on August 19, 1896, bound for Shanghai, with a crew of four officers and twenty-three men, and a cargo of kerosene. All the supplies were taken on board at New York. She touched nowhere on the voyage, and received no other supplies, except on one occasion fruit from natives who came alongside at the Solomon Islands, after the disease had broken out. When four and a half months out, at the end of December, 1896, the captain, a week later the first and second officers, and still five days later the third officer, son of the captain, found their legs, 'which had been numb for some time,' were swollen. They became rapidly weaker in the legs, and the first and second officers gave up work. 'Thirty-five days after the disease was first noticed, the first mate, and ten days later the second mate, died suddenly and unexpectedly, the former shortly after having his hair cut, the latter while eating, both displaying for several days before death great restlessness, palpitation, throbbing of vessels about head and neck, great difficulty of breathing, and both being perfectly clear-headed to the end.'

MacLeod saw the survivors in Shanghai on April 17, 1897, when they still presented some anæsthesia and loss of reflexes. His diagnosis of the disease, one with which he had been long familiar, was independently confirmed by another observer.

No member of the crew (of twenty-three) at any time showed any symptoms, nor did the Chinese cook nor the cabin steward. *It was confined to the officers, who messed together in the cabin.* All of them had left New York perfectly healthy. They 'took alcohol in no form.' Each had a cabin to himself.

'The first and second officers, when they were unable for duty, lived in the pilot-house on deck until they died, the captain and his son not changing their quarters during the voyage. When the captain first became sick, thinking he had indigestion before

¹ *British Medical Journal*, August 14, 1897.

the swelling appeared, he restricted himself entirely to maize-meal, stale bread, molasses, condensed milk, and hot water. He also put his son on this diet when his leg began to swell, and both took no other food until they reached Shanghai, while the first and second officers continued to eat of the usual cabin supply until they died.' (There can be little doubt that to this change of diet the former owed their lives.)

'The water-supply was the same for all on board. The crew messed forward, and consumed the same food as those aft, with the following exceptions: haddies, clams, oysters, lobsters, salmon (in tins), ham (not eaten by the captain), bacon, sago, tapioca, arrowroot, and boiled fruit. . . . *Rice* was consumed forward as well as aft.'

MacLeod, commenting on this carefully investigated case, well says that 'it points suspicion so strongly to a particular part of the food-supply, which comes from beri-beri-infested countries, as almost to amount to an actual experimental demonstration.'

They must certainly wait long who desire a more definite example of the production of beri-beri through food, clear proof of which, the reasonable must agree with MacLeod, this record of the *Ancona* supplies.

His view that one or another of the articles derived from beri-beri-infested countries was the source of the poisoning must also be accepted as correct, for were such things as tinned fish, etc., the produce of temperate climates, to be incriminated, the disease must necessarily be more frequently observed in countries using such articles, which is not the case.

The inquiry is thus limited to the articles sago, tapioca, arrowroot, and rice.

MacLeod seems inclined to exclude the last, on the ground that it was also consumed in the fore-castle. But it is to be observed, again, that whatever the source or vehicle of beri-beri on the *Ancona*, it must have been something common (or of a class or kind which was common) both to this ship and all the epidemics or cases of the same malady which occur elsewhere.

Now, of MacLeod's list, and the articles consumed habitually by the thousands of individuals in the Malay Peninsula who get beri-beri, not to mention numerous other instances, there is only one specific article or class of food in common—namely, the grain *rice*; for it can be confidently affirmed that neither sago, tapioca, nor arrowroot enters into the dietary either of Chinese coolies

in Malaya, the Japanese in Japan, or the prisoners in Eastern gaols, so often attacked with beri-beri.

It may be easily understood how rice may have been consumed in the fore-castle as well as in the cabin, and yet only have caused disease among the officers sharing the cabin mess, when it is considered that (as will appear more fully in these pages later), in poisoning by rice, as in other grain poisonings, it is the *quantity* taken which most determines the result. Those acquainted with the conditions of shipboard life and the prejudices of sailors will understand how little likely it is that the latter should have been fed often, still less systematically, on any sort of rice. Sailors would not accept rice instead of bread, or flour, or biscuit daily. Nor would they be likely often to get it offered them. Since, in the first place, it is, as a commodity of choice suitable for table use, usually much dearer, as retailed in Europe and America, than either wheat or maize meal; and, secondly, its preparation, in the only form in which it can be presented as a palatable article of diet—namely, in puddings made with milk, eggs, sugar, etc.—or in conjunction with curry, renders it a luxury which few ship-owners would be likely to find for their crews. The sailors on the *Ancona*, it may be presumed, therefore, got very little rice—perhaps a pudding on Sundays. On the other hand, the cabin-mess may have indulged in rice-pudding, rice and curry, rice as vegetable daily.

The *arrowroot* would no doubt be kept entirely for cabin use. It will surprise many to learn that the article thus named commercially, as made and sold by the best houses, is often nothing more than *pure rice-flour*. But Hassall¹ states this to be the case.

6. **Case of the 'Androcles.'**—MacMullen² narrates that in June, 1884, five seamen, forming part of the crew of the barque *Androcles*, were admitted into hospital in New Zealand, suffering from beri-beri. The ship left Amoy on February 4, 1884, carrying three Europeans (the captain and two mates), three Chinese (steward and two cooks), and eight native seamen. All the eight sailors, but none of the six Europeans and Chinese, were attacked.

'The men, when shipped, were allowed to choose whether they would have the same diet as Europeans, or that to which they had been accustomed. They chose the latter. . . . The Chinamen lived partly on the same food as the Europeans. The

¹ 'Food and its Adulterations,' 1876.

² *British Medical Journal*, November 21, 1885.

sick men ate well all the time.' The ship's melancholy log showed the entries :

' *March 10.*—Now ten days on salt provisions. Served out lime-juice and sugar; refused by several of the men.

' *May 5.*—Two men complain of having their legs swollen.

' *May 10.*—All hands except Europeans and Chinese have swollen legs.'

One of the Chinese had difficulty in passing urine, and swollen testicles (? beri-beric *œdema scroti* and bladder paresis), but recovered from this without further signs. He had not swollen legs.

Two of the patients died on board ship. Of the five brought into hospital, two were Japanese, two Malays, and one a Manila man.

Here again the dividing-line of incidence followed by the disease was one of diet, not of race. For three different races, all rice-eaters, suffered, while two races eating European diet escaped.

Evidence from Epidemics on Land.—On land, instances of beri-beri confined to rice-eaters only in the midst of communities, all the other (non-rice-eating) members of which should escape, are not so numerous, owing to the fact that the establishment of colonies of rice-eaters among nations of a different habit is itself not a common happening. But wherever such colonies have been established it has almost always sooner or later happened that beri-beri has appeared among them, and, what is the significant point, has invariably been confined to them.

7. **Epidemy in Manila.**—The earliest epidemy of importance, as to which the evidence recorded clearly shows the disease to have selected only rice-eaters, is that of which Königer¹ gives an account in 1884, as happening at Manila in 1882-83.

Beri-beri was unknown in the islands, Königer says, before this outbreak. Physicians of forty years' standing failed to recognise it. He himself, during three years of extensive practice at Manila, had not previously seen a single case. He was, however, well acquainted with its character, from experience in Japan.

In October, 1882, there was a great epidemy of cholera in Manila, to which from 15,000 to 20,000 persons fell victims in a population of 400,000. On October 20 a terrific cyclone devas-

¹ *Deut. Arch. f. Klin. Med.*, xxxiv., 1884.

tated both town and province, levelling all the lighter native dwellings with the ground; 60,000 families thus became suddenly bereft of shelter. Upon the cyclone followed great floods, which laid the low-lying parts of the town and neighbourhood under water, so that houses could not be rebuilt.

Then beri-beri appeared as a new plague, which took off its victims, after illness of a few days or weeks, in a mysterious and dreadful manner. Its march was widespread and rapid, and attended with enormous mortality, so that it was at first commonly believed to be invariably fatal. Not less than 60 per cent. of all cases, Königer thinks, died in these earlier days of the epidemic. As regards the case-incidence, no reliable figures could be given; but in one district alone—Malabon, where there were 25,000 inhabitants—300 deaths from beri-beri took place in November and December, or 1.2 per cent. of the whole population died in two months. He estimated the whole tale of victims at *many thousands*.

Königer, reviewing all the circumstances under which the epidemic appeared, notes that the population of Manila consisted of three principal races, between whose habits of diet there were great differences. There were the Europeans (from 7,000 to 8,000 in number), whose diet, generous and very varied, contained always more meat than is customary even with Germans in Europe; next the Chinese (5,000 of the richer trading classes), who, in addition to rice, ate plenty of vegetables and of meat; last, the natives (50,000 Filipinos or Manila Malays), whose 'diet, year in and year out, is dry rice, cooked in water, with a little fish, perhaps some fruit, and a few vegetables.'

Now, during the cholera epidemic these natives had been wholly deprived of *fish*, for they feared to take them from the rivers and canals, which were polluted with cholera excreta, while the sea-fisheries, never considerable, were at that time of the year closed by the monsoon.

Of fruit, at the same time, there was none to be got. Even the richer half-castes, priests, and others, who subsequently got beri-beri, then ate (they informed Königer later), for fear of the cholera, for whole months nothing else than rice.

When beri-beri appeared, it was these natives (and exceptionally Chinese and Europeans who, like them, were living wholly on rice) who alone were attacked.

Among the large European colony, Königer heard only of two cases of beri-beri—he himself met with none—and these two

were destitute paupers, who were probably eating rice like the natives. Among the Chinese, well-to-do, and able to buy flesh, eggs, fowls, etc. (their usual diet), though at dearer prices, cases were extremely rare.

Königer adds that among a great number of cases of *anæmia* under his treatment, put upon a special meat diet, not a single case of beri-beri happened, although they were at some of the worst centres of the disease; and he notes especially that native domestics in European employ who partook of the same food as their masters never suffered. A single apparent exception to the last rule turned out to be a case of chronic nephritis.

In this epidemic women and men were equally attacked, and there was no discrimination of age, except that very young children seemed to be spared. To women lying-in it was especially fatal.

Königer himself does not attribute beri-beri to any poisonous effect of rice. His view seems to be that the disease (which he regards apparently as an infection) selected especially the natives, as the result of their having become *predisposed* to it, through the combined action (*zusammenwirken*) of the several calamities to which they had been subjected.

No doubt what weighed with Königer against the probability of the disease being directly due to the rice was the fact that the natives affected were always, before as after the epidemic, rice-eaters.

Perhaps this outbreak should rather have been placed among those instances of beri-beri which are to be shown to depend upon the consumption either of a greater *quantity* or of a different *sort* of rice than ordinary.

It is certain that, as a result of the cholera, and the cyclone, and the flood, the natives, deprived of the usual adjuncts—fish, fruit, vegetables—by which they were wont to supplement their diet, were reduced to living on *rice only*, and so to eating it in excess, as compared with their custom in ordinary times. It seems likely also that the two later calamities may have effected great or even total destruction of their stores of padi, garnered always by natives under their own houses. These, even if not altogether borne away by wind or flood, may have been soaked and ruined by water. Under such circumstances, they would be reduced to procuring rice from other quarters—*i.e.*, would have recourse to shop (white stale uncured) rice—consumption

of which, it will be shown later, more than any other sort induces beri-beri.

It was this combination of circumstances—*i.e.*, of excess in quantity, and use of a noxious sort (stale as compared with fresh rice)—which, far more probably than that of depressing influences, as Königer supposes, wrought to produce the unique and terrible epidemic of beri-beri in Manila. For such calamities have indeed happened elsewhere, but never with like effect. Nor, indeed, is it conceivable how, regarding beri-beri as due to an *infection*, any amount of predisposition can effect the *introduction* of the disease into a community among whom it has never before been known, still less, when it has appeared, limit its attacks to individuals only of a particular nationality.

But here is submitted, what the clear and unprejudiced statement of facts given by Königer makes evident: that in the great epidemic of beri-beri at Manila the incidence of the disease was *solely upon rice-eaters*; all those who ate no rice (as also most of those who ate but little of it) escaped.

8. **American Troops in the Philippines.**—I have said above, writing in 1902, that since this great epidemic beri-beri had never reappeared in Manila. History has, however, repeated itself with unusual exactitude of detail. Smart¹ records epidemics occurring, 'prevailing extensively among the native troops, at Laguan, Samar, in August, 1902; in Iloilo in September and October; and at Cauayan, Isabela, in 1903.

'The 39th and 44th companies of Philippine Scouts were the sufferers at Iloilo. The commanding officer of the latter company states that for a year the health of his men was excellent, and no case of beri-beri occurred among them. *Owing to the prevalence of cholera in the neighbourhood, communication with the markets was interdicted, and this quarantine cut off the supply of vegetables, restricting the command to a diet of rice and meat. In the third week of the quarantine the first case of beri-beri occurred,* and soon afterwards one-third of the company was affected. The italics are the present writer's.

Smart himself, it must be added, suggests that 'the disease was possibly not caused by the rice, but by the want of fresh vegetables that had heretofore been associated with it'—a view already discounted. He goes on to say that 'rice, if a factor at all in the production of the disease, is less powerful than a specific infection of locality'; and he notes, in giving account

¹ *Rep. Surg.-Gen. Army, U.S.A., 1902-1903, p. 69.*

of the 'latest endemic,' in March, 1903, that those affected 'were getting the full native rations, and drew flour in preference to rice.' The objection to rice necessarily raised by this observation will be discussed later on. The flour probably was not eaten.

During the fiscal year 1902-1903, among the United States troops, O'Reilly states¹ beri-beri 'caused 626 admissions, equal to 7.75 per 1,000 of strength, a rate for the whole army larger than that of typhoid fever. All cases originated in the Pacific Islands, and nearly all among Philippine Scouts. There were 23 cases and 2 deaths among white troops, 5 cases with 1 death among coloured, and 598 cases with 29 deaths among Filipino soldiers.'

In the following year the same state of affairs continued. 'Beri-beri was almost entirely confined to the Filipino soldiers, who gave an admission-rate of 1128.21 per 1,000 men, 4.59 in every 1,000 terminating fatally. There were three deaths from this cause among white troops.'²

Yet 'in the diseases of the nervous system'—beri-beri being excluded—'the Filipino troops show a rate of admission per 1,000 equal to about one-third of the rate reported for white troops, who had 24.57 men out of every 1,000 admitted for these affections, while coloured troops to the extent of 19.79 per 1,000 were admitted to sick report for the same causes. The latter led the white troops in mortality-rate from nervous diseases, while the Filipino soldiers lost 0.42 men per 1,000 of strength—a rate slightly in excess of either that for the white or coloured troops.'

There were no coloured troops on duty in the Philippine Islands during this year.

The table following compares the relative case-incidence of beri-beri on the three races composing the American Army in the Philippines for the two years mentioned :

Year.		White.	Coloured.	Malays.
		Strength. 30,000	Strength. 2,642	Strength. 4,826
1902-03	Admissions for beri-beri per 1,000 of strength	0.71	0.18	123.92
		Strength. 18,930	Strength. —	Strength. 4,888
1903-04	Admissions for beri-beri per 1,000 of strength	1.10	—	128.21

¹ *Rep. S. G. A.*, 1902-1903, p. 69.

² *Rep. S. G. A.*, 1903-1904, p. 58.

The exemption of the coloured troops, greater in proportion even than that of the whites, under circumstances where it is certain that they could have enjoyed no hygienic advantages not shared by other native troops, is all the more significant. For the only difference between them and other natives who were prone to beri-beri lay in their food. They, like the Europeans, are wheat-eaters, or at least eaters of other things—maize-corn—than rice; while the latter grain is the staple of the Filipino.

Coloured races—negroes—suffer, however, sufficiently severely elsewhere. Prout¹ has mentioned an epidemic among negroes returning from the West Indies. Bourguignon² states that on the Congo 'Barbadian negroes . . . , men from the Guinea Coast, succumbed by thousands to paludism and beri-beri.' Lasnet³ noted cases in the prison at Dakar. Negro troops—the Senegal Rifles—furnished the chief number of beri-berics in the epidemics described by Schuttelaere⁴ at Diego Suarez in Madagascar. But in all these places their diet had been chiefly rice.

9. European Troops in the Dutch East Indies and Elsewhere.—The susceptibility of the European to beri-beri, which is no less, again, than that of any other race, when the necessary conditions are brought to bear, though generally admitted, is seldom realized. The fact is, however, one upon which insistence is important, because the exemption of Europeans from beri-beri in places whose other inhabitants are prone to it, becomes thereby evidence of value.

How great the liability of Europeans to the disease may be can best be gauged by a consideration of its incidence upon the troops of their own race employed by the Dutch in their East Indian possessions—Java, Madoera, and Sumatra.

During twenty-two years of occupation—1873 to 1894—it appears from figures furnished by Van der Burg⁵ that the average strength of European troops employed was 15,200 men. In that term there occurred in the force 17,520 cases of beri-beri. The natives fared far worse.

For the first eleven years the incidence on the Europeans had

¹ Prout, W. T., *Proceedings British Medical Association*, July 30, 1902; *Lancet*, 1902, vol. ii., p. 395.

² A. Bourguignon, G. Dreypondt, and Ch. Firket, 'Congrès Nat. d'hygiène et de Climat. Méd. de la Belgique et du Congo,' 1897, part ii., p. 472.

³ Lasnet, *Arch. ins. de Méd. Nav. & Hyg. et Cos.*, February, 1897, p. 138.

⁴ *Arch. de Méd. et de Pharmacie Militaire*, t. xxxvii., No. 12, December, 1901, p. 470.

⁵ *Weekblad van het Nederl. Tijdschr. v. Geneesk.*, No. 3, January, 1896.

been mild—no more than 4, 5, or 6 per 1,000 of strength. In 1883 it rose to 15, in 1885 it was 122, and in 1886—the severest year—258 per 1,000.

The three succeeding years saw great decline ; but since that date down to 1894 the rate had never fallen below 71 per 1,000 of strength.

Now, while the conditions under which the Dutch troops have had to live in their colonies differ, as far as external factors go, in no wise from those which apply to Europeans in other tropical areas, including places such as British Malaya and Cochin China, whence the disease is never absent, there was (and is) between the Dutch in their colonies and European dwellers in other tropical places the single, almost constant, and, as will appear later, the all-important difference that the former eat a different sort of food. The Dutch almost everywhere in their colonies acquire the habit of eating rice largely, and a large part of the rations of their European troops, both in cantonment and on active service, is rice. Davidson states that the disease attacks men on active service far less severely than those in barracks, an experience which seems to have met with corroboration under other circumstances. In the case of the Dutch troops this lessened incidence is explicable by the fact that the *proportion of rice* in the diet is less when on active service. I find, from an official account, that the proportion of rice in the diets of the Europeans when in garrison is 42 per cent. of the whole ration, and in that of the natives 72 per cent. When upon 'excursions'—expeditions—lasting over two days, this proportion is reduced—in the case of Europeans to 21 per cent., and for natives to 55 per cent. On the other hand, Overbeck de Meijer (says Hirsch) states that 'whenever the troops engaged upon military expeditions into certain parts of the Dutch East Indies are obliged to live *exclusively* on the ordinary food of the country, they are almost *always attacked* by beri-beri.' Pop, Hirsch says, confirms this.

The disastrous incidence of beri-beri on these European rice-eating troops must be compared with the almost entire immunity of other European but not rice-fed troops in other countries which are centres or 'endemic haunts' of the disease, no less notable than the Dutch East Indies. The French in Africa, Cochin China, Tonkin, and Madagascar, the Belgians on the Congo, the Germans in New Guinea, the British in Burmah and the Straits Settlements, furnish sufficient examples to justify the

statement that troops employed in numbers, however large, and for whatever period of time, do not get beri-beri even in places where the other inhabitants do so, so long as they are not fed upon rice.¹

Similarly with civilian populations. Not less do the Dutch experiences—the severe incidence of beri-beri upon rice-eating Europeans in their colonies—explain and lend force to the remarkable exemption of civil communities of Europeans who are not rice-eaters from beri-beri, in other countries whose native, rice-eating inhabitants are perpetually scourged by it.

Of such there are many and conspicuous examples.

10. Europeans in Kaiser Wilhelm's Land.—Wendland² has thus noted the complete freedom from beri-beri of all the Europeans employed in German New Guinea during the course of an epidemic which he witnessed there in 1895. Every class of native—Chinese, Javanese, Melanesians, Jabims (the aborigines)—he states, suffered, but the Europeans enjoyed complete protection—*vollige immunität*.

11. Europeans and Ainhos in Yezo.—Grimm, whose experiences were gained in this, the cold northernmost island of Japan, says³ that there both Europeans and Americans were always exempt; so also were the Ainhos—the wild aboriginal inhabitants of the country—so long as they adhered all of them to their own customs in regard to food.

¹ To the rule thus broadly stated it is, of course, likely that apparent exceptions may occur. Among troops located in rice-eating communities it will almost certainly happen that a few individuals will acquire a habit of eating rice in quantity. Unless their habits were closely investigated, persons who should get beri-beri under such circumstances would be likely to be put down as cases of the diseases not derived from rice. Then there are the many forms of peripheral neuritis due to other causes than that of beri-beri, but which are yet undistinguishable from the latter malady, and so may always be a source of confusion.

A few such cases happened in 1900 and 1901 among the British military stationed at Mandalay, Meiktila, and Shwebo in Burmah. The reports of these cases were placed at the writer's disposal by the kindness of the Principal Medical Officer Madras Command, Major-General Sinclair. The cases, some twenty-two in number, were all, with one exception, very slight, and the very careful descriptions of them furnished by Fairrie, Nichol, and Thomson, who diagnosed them as beri-beri, left the impression that they were, at all events, not typical examples of the complaint. In none of them was rice excluded as the possible cause.

² *Arch. f. Schiff's u. Tropen Hyg.*, 1897, Bd. 1., H. 4, p. 237.

³ F. Grimm, *Klin. Beobach. über*, 'Beri-Beri,' 1897, p. 120.

When Europeans adopted Japanese habits, he says, they got beri-beri. When the Ainhos, who in their wild state subsisted chiefly on fruit, fish, and other wild products, came to mingle with and live like Japanese, they, too, became liable to it equally with the latter. Grimm, who is no believer in rice as the cause or vehicle of the disease, attributes this result to differences in the preparation of the food. But it seems clear that the common factor determining the exemption of these two races—who had absolutely no other feature in common—was the fact that neither of them ate rice.

That which determined their liability to the malady, when they did get it, was the adoption of rice as their staple.

12. Europeans and Americans in South Japan.—In Japan beri-beri has been known (as kakké) from remote ages, and it is more severely prevalent there to-day than perhaps in any other country.

There has long been a considerable white population—Europeans and Americans—settled in the southern islands. These people form a community whose members, in trade and socially, and in their domestic arrangements have long been in close and intimate association with the natives of the country. Were beri-beri anything communicable from person to person, or from places to susceptible persons, it is impossible that, prevailing as it does largely among the one class, it should not also spread to the other. The disease chiefly occurs, says Saneyoshi, in the large towns and seaports. It is at these places that the Europeans principally reside. It attacks members of well-to-do classes even oftener than the poor. In these classes are the Americans and Europeans. Everything, therefore, in the external factors should favour the white immigrants being attacked by beri-beri.

But this does not happen. Instead, the Americans and Europeans in Japan have enjoyed an immunity from the disease which has been continuous and all but mathematically complete.

Anderson notes that not a single case of beri-beri happened among either the French or British troops. Simmons saw but a single case in a European. Scheube had seen but two. Baelz mentions none.

I have been able to glean no information as to what were the habits in regard to food of those exceptional individuals among the Europeans who did get beri-beri. But since that factor has not been excluded in their case, it is to be supposed that

they had taken to rice-eating, and thereby became, for the purpose of comparison, ranged with the Japanese. For it has to be formally stated: between the Japanese, perpetually afflicted by beri-beri, and the many equally susceptible Europeans who remain, while in Japan, as perpetually exempt from the malady, there is this single important difference: that the Japanese are rice-eaters, the Europeans are not.

The above and some instances to come exemplify the immunity of non-rice-eaters from beri-beri in places where rice-eaters are attacked.

Supplementing them and completing the conviction they afford, are many instances where the circumstances have been inverted yet the same result has been obtained.

Rice-eaters, newly immigrant into places where beri-beri has never previously been known, have incurred the disease, and have alone been attacked by it, while the native residents, not being rice-eaters, have remained exempt.

13. **Epidemics in New Caledonia.**—The islands called by this name were adopted by the French as a penal settlement in 1859.

There were 21,630 convicts (*forçats transportés*) conveyed thither between this date and November 28, 1896, after which no more were sent. Of these, 1,713 only were 'natives'—Africans, Asiatics, or Polynesians—the rest were European.

In addition to the convicts, after 1885 there were sent there annually several hundred *relégués*—banished recidivists.

From 1880 onwards there was thus an average European population in these islands of over 8,000 persons.

The climate, a mild and equable one, permitted them manual labour. They were fed on the same sort of food-stuffs as in Europe.

The mortality was, though high, not severe. During the thirty-three years 7,222 of the convicts—about one-third—had died.

Kermorgant,¹ from whose interesting account these particulars are taken, states that the principal causes of death were: diarrhœa and dysentery, over a quarter; tuberculosis, enterica, about one-tenth of all deaths each; chest troubles and anæmia, each about 5 per cent. of the deaths. There was no malaria.

¹ A. Kermorgant, 'Morbidity et mortalité des transportés et des relégués en Guyane et en Nouvelle Calédonie,' *Ann. d'Hyg. et de Méd. Col.*, t. 6^{me}, No. 1, p. 153, January, 1903.

110 THE CAUSE AND PREVENTION OF BERI-BERI

Beri-beri is not mentioned, and it must be presumed did not occur. It is, in fact, stated by Grall, Vincent and Porée, that before the event to be described, this disease had never been known in the islands. Here, then, was a population of non-rice-eating Europeans, living otherwise (as convicts) not under the best hygienic conditions, among whom beri-beri had been, from 1858 to 1891 at least, unknown.

In the latter year were introduced into the islands some 800 Annamites and Tonkinese, convicts from Cochin China.

These men were fed almost exclusively on rice. Beri-beri broke out among them immediately after their arrival. Two hundred were severely—all perhaps slightly—attacked, and seventy died. At the height of the epidemic these people were distributed from the quarantine island, where they had hitherto been kept, among several stations. There was thus opportunity given for the malady—were it infectious—to spread. But no Europeans were attacked.

In 1892, 600 healthy Japanese recruited as labourers were brought to the same settlements. They, too, were fed almost wholly on rice. They, also, shortly after arrival got very severe beri-beri. Two-thirds are recorded as having been attacked, and there were many deaths.

But again the disease remained entirely confined to the rice-eaters. Not a single European acquired it.¹

Ten years later, Judet de la Combe² relates, several hundred coolies were again brought from Japan to work in the nickel-mines. They were inspected and passed as healthy by a medical officer before embarkation.

Their diet was : the best Japanese rice, 1 kilo per head daily ;

¹ *Arch. de Méd. Nav.*, t. lxiii., Nos. 2, 3, 4, pp. 134, 187, 260, February-April, 1895 ; also Hagen, *Rev. Méd. de l'Est*, t. xxv., No. 2, p. 42, January 15, 1893. It was from a consideration of facts connected with the epidemic among the Tonkinese that Hagen, against most authorities and all the facts, announced beri-beri to be contagious. Certain Polynesian labourers under Hagen's own care developed, after the outbreak among the Tonkinese, symptoms resembling beri-beri, and diagnosed by Hagen and Kieffer, the chief local medical officer, to be beri-beri. There had been opportunity of infection between the two classes. Without discussing the likelihood, always highly probable, that both these lots of labourers had shared the same sort of food, Hagen concludes that his coolies must have incurred the disease by direct contagion. He does not even mention the fact—which, however, Legrand and Burot supply (*Maladies des Marins*, etc., 1896, p. 86, footnote)—that the Polynesians in question were like all who then got (or get) beri-beri, being also fed almost wholly upon rice.

² *Ann. d'Hyg. et de Méd. Col.*, t. vii., No. 3, p. 326, July-September, 1904.

fresh meat, 250 grammes; salt fish, 90 grammes; pulse, 70 grammes dry, or 250 fresh.

Upon this diet beri-beri was severe. In 1901, with a force of 618, there were 489 cases.

In 1902, with a force of 328, there were only 8 cases; and in 1902, the average strength being 246, there were 28.

The remarkable reduction in 1902 followed upon a reduction in the amount of rice, with an increase in the quantities of meat and fat, and will be further referred to later in this work.

Although nothing is said upon the point, it may be taken for certain that this epidemic remained confined, as before, to the eaters of rice, and that no Europeans suffered.

14. *Japanese in Fiji.*—In the Fiji Islands, prior to the event now to be described, beri-beri had never been known. The aborigines—Polynesians—use for their staple food *taro* (yams), also corn (maize), and other vegetables, and they eat much fish. European and other settlers eat bread made from wheat-flour as elsewhere.

'In 1894¹ a number of Japanese were imported into Fiji to work on sugar estates. They were picked coolies, and in good health when they left Japan, and on the journey. On their arrival at Fiji they were divided into two bands. One, consisting of fifty coolies, was sent to work on a certain estate. The dietary was liberal, including meat or fresh fish to the amount of $\frac{1}{2}$ pound per diem, besides 2 pounds of rice and a variety of condiments.

'Nevertheless, within two months of the arrival of the coolies on the island, beri-beri broke out, and in all 42 cases occurred. Eight of these patients died; 34 were returned to Japan suffering from the disease, the remaining 8 healthy coolies accompanying them. The other batch of immigrants—205 in number—were sent to a sugar estate in another part of the island. They had a similar dietary. Within a month beri-beri broke out among them.

'The cases steadily increased, and by the end of six months 226 out of the 250 were affected; 69 died. The survivors—181 in number—many still affected with beri-beri, were sent back to Japan.'

I have been favoured by Dr. Glanvill Corney, the present

¹ I quote this succinct account of the circumstance from Manson's article in the *Lancet*, November 24, 1901, vol. ii., p. 1393, already referred to. A fuller account is given by Joynt, *Journal of Tropical Medicine*, May, 1, 1901, and by Corney, Hirsch, and Joynt, *Report Leg. Council Fiji*, 1896.

Chief Medical Officer of Fiji, with some further interesting particulars relative to these ill-fated Japanese.

Thus, the quarters occupied by them 'were newly built specially for them to occupy on arrival ; and it was a condition of their contract that for the first six months' residence all their rations should be imported from Japan. Later, however, they were given rice which came from Calcutta, and was of the common stock issued to the Indian coolies and native labourers on the same company's plantations. . . . The Japs did not work in the same field batches as the Indian coolies or Fijians.

'There are no Chinese coolies in Fiji. But after working hours there was absolutely no bar, except social differences, between the Japs and all other nationalities on the estates or in the surrounding native villages, and it was an everyday occurrence for them to stroll about together. . . .

'The rice imported from Calcutta was ordinary Ballam rice, such as we use in Fiji year after year.

'No person in Fiji has had beri-beri except the Japs, who came on the occasion referred to, and, in 1904, a couple of newly-arrived Chinese merchants' employés. These two men never went to either of the districts where the Japanese were located in 1895-1896.'

Here was an epidemic, the cause of which, were it an infection, was at once virulent and active enough, and placed under apparently perfect conditions for spread. Its victims were many and severely stricken ; they were in constant intercourse from the earliest to the last stages of their malady with classes of natives whom experience elsewhere has shown to be peculiarly susceptible. For Indians have suffered severely in Malayan gaols, and Polynesians are the very class of natives whom Hagen claims to have succumbed to beri-beri in New Caledonia through mere chance and quite brief contact.

Yet the disease did not so spread. It was confined to those who were rice-eaters, and among these to the eaters of one particular sort.

The immunity of those rice-eaters who were fed on Ballam rice during this epidemic is also a fact of very great importance, the significance of which will appear later on.

Such instances as the above have been multiplied in almost all parts of the world wherever parties of the indefatigable nation have settled. Like the Nessian shirt, their use of a certain sort of rice, wherever they have gone, has been an inalienable secret factor leading to destruction.

Saneyoshi¹ mentions the following instances :

15. At a station, **Okinawa**, in the Loochoo Islands, the Japanese soldiers constantly incurred beri-beri. They were garrisoned on the highest and healthiest spot in the island, and the natives of the island themselves had never been known to have beri-beri. This was before the introduction of the new regulation diet. The rations of the Japanese then consisted principally (56 per cent.) of rice. But the natives, 'owing to a scanty production of rice and barley, live on peas and other grains and pork.'

16. 'In **Korea**,' Saneyoshi observes, 'the Japanese and Koreans live side by side. The Japanese commonly have beri-beri, while it occurs only rarely among the Koreans. The Korean houses are small and dirty, but the inhabitants feed chiefly on peas, and do not eat rice as the Japanese do.'

17. Beri-beri is not prevalent among the inhabitants of **Van-couver**, but Bailey reports an epidemic among Japanese in a small settlement on the island. 'His observations favour the dietetic origin, a *too exclusive diet* of rice being conducive to the condition.'²

17. **American Rice-eaters in U.S.A.**—In the United States of America cases of beri-beri from time to time are received in the hospitals off ships. It has not hitherto been supposed to be endemic anywhere, but Young³ has recorded cases occurring at Abbeville in Louisiana, which must have originated there. During the last five years Young states he has met with 'many cases' of beri-beri, and he mentions three other physicians who had recently treated thirty, six, and six cases of beri-beri each, and all agreed as to the diagnosis. The cases all came under observation within a period of twenty-four months, and within an area of twenty miles radius.

Young further states that in the last fifteen years the culture of *rice* in his district had developed enormously. 'I have been practising in this parish for twenty-five years, and up to five years ago never saw a case of beri-beri, nor anything resembling it. . . . Since that time there have been in this district no less than forty cases of a disease we have termed beri-beri. . . . The cases have occurred without exception in a locality surrounded

¹ *Sei-i-kwai*, April 30 and May 31, 1901.

² J. W. Bailey, 'A Clinical Study of Beri-beri.' Reference : *Journal of the American Medical Association*, 1903, vol. i., p. 1324.

³ F. F. Young, M.D., *Journal of the American Medical Association*, 1903, vol. i., p. 111.

by rice-fields, and *all the patients were heavy eaters of rice* (the present writer's italics).

18. **Cases amongst the Chinese in Australia and Calcutta.**—In Australia small colonies of Chinese are settled here and there, living after their manner, crowded in single tenements in towns. The food they eat is rice. It is only among these small rice-eating groups that beri-beri has been known to occur, upon which very insufficient ground the disease has been referred to as 'endemic' in Australia by more than one observer.

The *British Medical Journal* of September 28, 1895, referring to accounts of such cases in Melbourne and Sydney by Graham,¹ Paton,² Corlette,³ and others, has adopted this view.

Commenting on Corlette's account, this journal says: 'Hitherto only Chinese and other Orientals have been attacked. . . . Moreover, it was ascertained that the disease tended to occur in little epidemics, several cases coming from one house, a fact distinctly pointing to the existence of local endemic foci.' But this fact would equally consist with food-poisoning, the cases of which naturally run also in groups. To dub a malady 'endemic' in any country merely upon such showing as this is singularly to attenuate the meaning of the word.

No one who has any experience of Chinese or other Orientals can doubt that in Australia, as elsewhere, they adhere tenaciously to the diet to which they are accustomed, and that the cause of the disease in these small colonies of immigrants lay in the rice which they themselves imported.

Corlette⁴ describes a single case in a European in Australia—one child in a family, none of the other members of which were attacked. He attributes it to infection from playing in a shed, a previous occupant of which had been a Chinese gardener, who does not appear to have himself had beri-beri, and from playing with Chinese neighbours, none of whom had beri-beri either! To such an extent do the probabilities become strained to demonstrate origin of the disease by infection! Nothing is more likely than that a child playing about among Chinese rice-eaters, however, should have frequently eaten of their rice.

Beri-beri has become of late years increasingly prevalent in Australia, even among whites. It is among squatters and their hands living far up-country that these cases occur. At these stations, though fresh meat is plentiful, fruit and fresh vegetables are often scarce, or not obtainable. They are then often replaced by rice, which is regarded by some as an efficient substitute for them, and even as an antiscorbutic.

One who had himself suffered from severe beri-beri acquired in Australia under these conditions informed the writer that for a whole year rice for vegetable, rice and curry, rice-pudding, was, except on Sundays, part of his regular daily fare.

¹ *Austr. Med. Gaz.*, November 15, 1893.

² *Ibid.*, November, 1895.

³ *British Medical Journal*, 1895, vol. ii., p. 800.

⁴ *Ibid.*, 1897, vol. ii., p. 680.

In Calcutta, where the great majority of the natives do not eat rice, but millet, wheat, corn, and other grain, it is again among the small Chinese colony of artisans, petty shopkeepers, etc., Buchanan¹ says, that cases of beri-beri are almost solely found.

19. Incidence in the Indian Native Army.—In the Indian Army the special liability of the Madras troops to get beri-beri, as compared with other arms, has long been noted, and yet remains.

Malcolmsen, in his 'Practical Essay' (1835), observed that 'the Bengal sepoy, who shuns animal food and is very parsimonious, and far more sickly than the Madras, yet is not subject to beri-beri,' and that the difference between the two was that 'the Bengali sepoy eats sparingly of wheat, and never any rice.'

Through the kindness of the principal medical officer Madras command, Major-General D. Sinclair, I have been permitted to peruse several reports upon outbreaks of beri-beri which have occurred among Madras regiments in recent times.

During the years 1896-1901 epidemics of beri-beri have happened among the troops stationed at three places only—viz., Vizianagram (successive years 1896-1901), Rangoon (1899-1901), and Trincomalee (1901). All of these are places at which the disease was known to occur among the natives. Details of the troops concerned and the number of cases appear as under :

Station.	Year.	Regiment.	Cases.		
			Men.	Recruits.	Total.
Vizianagram ...	1896	20th M.I.	12	43	55
" ...	1897	20th M.I.	—	—	—
" ...	1898	2nd M.I.	31	42	73
" ...	1899	2nd M.I.	66	47	113
" ...	1900	— M.I.	—	—	11
" ...	1901	26th M.I.	—	—	15
Rangoon ...	1899	7th M.I.	—	—	36
" ...	1900	7th M.I.	—	—	12
" ...	1901	7th M.I.	—	—	15
Trincomalee ...	1900	{ 9th M.I. }	—	—	9
" ...	1901	{ detachment of 138 men }	—	—	36

The daily ration of Indian troops, substantially the same everywhere and for all arms, is as follows :

¹ *Lancet*, August 27, 1898, vol. ii., p. 577.

116 THE CAUSE AND PREVENTION OF BERI-BERI

Rice or attah ¹	2 lb.
Meat ²	5 oz.
Dhal ³	2½ oz.
Ghi ⁴	¾ oz.
Fresh vegetables	6 oz.
Various adjuncts ⁵	6½ oz.

By the Madras rice is drawn in preference to flour, and it thus forms 61 per cent. of whole ration.

Provisions are always bought locally in the bazaar of the station at which the troops are quartered. Hence the rice used is always the same as that consumed by the villagers of the neighbourhood, and in the case of most of the districts of the Madras Presidency, at least, the sort of rice eaten is not made from padi boiled previously to milling. That is to say, it is the sort which will later on be described as 'uncured' rice. In Rangoon the rice sold in the shops is for the most part the same. So also in Ceylon.

Several most significant facts appear in the reports of these epidemics which the writer has been privileged to read. Among them the extraordinary liability to the disease of *recruits* as compared with *men*, and certain fluctuations in the epidemics observed to be coincident with alterations in the rice supplied. These will be dealt with later.

Here the fact only is emphasized that among all the troops of the Indian Army the only sufferers from beri-beri are—have for generations been—those whose staple of food is rice.

Naturally, the liability to beri-beri which their rice diet entails upon Madras troops follows them out of India.

Thus, fifty-two men of the 42nd were attacked, Manson⁶ says, when on service in China in 1847. Similar cases happened in the recent occupation, by different Allied troops, of Peking.

At Singapore cases have from time to time happened among rice-eating native troops in garrison, but never among the Europeans.

The most striking of such examples is, perhaps, the following:

Cases amongst Madras Troops in the Soudan.—Beri-beri is not endemic in the Soudan. The chief food of the natives is millet, maize, and wheat; rice is a luxury. References as to its occur-

¹ Wheat-flour.

² Mutton or fowl.

³ Pulse, husked and split.

⁴ Native butter or lard.

⁵ Including onions, ½ oz.; tamarinds, 1 oz.; salt, 1 oz.; curry stuff, 9 drachms; pepper, 1 drachm; garlic, ½ drachm.

⁶ Davidson, *Hyg. and Dis. Warm Climates*, p. 454.

rence in that exceptionally hot and dry region have escaped even Hirsch's industry.¹

But Smyth related² in 1888 that 'during the last expedition in the Soudan the Madras sappers and miners on service there suffered severely from beri-beri, while not a case occurred in any other arm of the service. *They were the only troops present with whom rice formed the staple article of diet.* The rice, however, was of fair quality, and was used constantly by the European officers without any bad effect.'

The latter part of this statement, of course, in no way prejudices the view that rice caused the disease in the natives. The inference it allows is that in noxious rice, as in other poisons, the result is proportionate to the quantity taken. An occasional meal will not produce beri-beri, though persistent dieting on the poisonous rice will do so.

This instance stands as evidence of the strongest in favour of beri-beri depending on rice. Whatever way the circumstances of soldiers on an expedition be considered, it seems impossible to conceive the limitation of an infectious disease to those who took a particular sort of rice only, and a causal connection between the rice and the disease in this instance seems impossible to explain away.

The limitation of beri-beri historically as an endemic disorder of India principally to the Madras district, the Northern Circars, Ceylon, and Burmah, itself points to rice as the etiological factor; for, regarding India as a whole, as a community of States, it may be said that the regions referred to are the only ones among them in which rice is the chief cereal grown, and the staple food of the people.

Evidence from the Malay Gaols and Asylums.—Instances yet more striking are to be recorded of the singular and persistent restriction of beri-beri in institutions to those members of them only who eat rice, although all are in constant and close association, and none are distinguished by the operation of any special condition.

These are furnished by the gaols and asylums of the Straits Settlements and native Malay States.

20. In the **Singapore Lunatic Asylum** patients of every nation-

¹ Christopherson has since reported cases also in rice-eaters (*Journal of Tropical Medicine*, May 15, 1903).

² At a meeting of the South Indian and Madras Branch of the B. M. A., May 18, 1888 (*British Medical Journal*, January 26, 1889, p. 193).

118 THE CAUSE AND PREVENTION OF BERI-BERI

ality are received, the daily average number of patients resident being usually about 200.

In 1898 there were 173 admissions—12 Europeans and Eurasians, 116 Chinese, 12 Malay and allied races, 22 Tamils, 6 Bengalis and Sikhs, and 5 of other nationalities. In 1900 the number of admissions was 165, composed as follows: Europeans and Eurasians, 16; Chinese, 105; Malays and other races of the Archipelago, 10; Tamils, 24; Bengalis and Sikhs, 4; other nationalities, 6.

I have not been able to obtain the races for every year, but the composition of the asylum population is usually much the same. In addition to the patients, a large number of native warders of different races, including Chinese, Javanese, Sikhs, and Bengalis, reside at the asylum. The institution has been severely visited by beri-beri—so much so that the superintendent, Dr. W. G. Ellis, writing in 1897, says that ‘the asylum must now be looked upon as a pest-hole of beri-beri’¹—a state of affairs which, it seems, has unfortunately continued ever since,² as the following table will show:

Year.	Total Admissions.	Average Daily Population.	Number of Cases of Beri-beri treated.	Deaths from Beri-beri.	Deaths from all Causes.
1896	?	223	?	40	85
1897	402	228	667	48	81
1898	382	208	201	55	95
1899	335	184	121	29	65
1900	365	180	153	51	123
1901	340	164	110	15	56
1902	331	174	99	4	42
1903	400	209	18	3	55

The total of 667 beri-berics for 1897 was obtained by adding together the total cases under treatment during each month for the whole year. But in 1898 Dr. Ellis says the table given ‘shows the number of patients attacked month by month during the year. . . . Under no circumstances has the same patient been counted twice, though relapses are frequent; perhaps one should say further attacks, for many cases have intervals of months of good health.’

It is evident that here, again, were ideal conditions for studying beri-beri—a confined community, well housed, well fed, under strict control, under medical supervision, of all nationalities, and for the most part under observation for long periods of time.

¹ *Ann. Med. Rep. S.S.*, 1898, p. 13, par. 12.

² This was penned in 1902. In spring, 1903, the disease abruptly ceased; but 14 cases occurred in the last two months of the year.

Were the cause of beri-beri anything of the nature of an infection, it must have ensued that all the nationalities should be equally attacked; for it is incredible that, in the confined space of an asylum, patients of any particular race could have wholly escaped infection by a disease which was not merely prevalent, but pestilentially so, and that for a period extending over many years.

But the incidence was not equal.

In his report, 1898,¹ Dr. Ellis writes: 'Seven attendants suffered from beri-beri, two of whom had to leave the service on account of persistently recurring attacks.'

In 1899 he remarks:² 'Attendant Sangei, a Javanese, had to leave on account of suffering from chronic beri-beri, and Attendant Lee Hoe, a Chinese, was granted six months' leave for the same reason. It is curious that *we have never yet had a Bengali attendant (we have eighteen of them) attacked with the disease, nor have any of our European or Eurasian patients ever suffered*' (writer's italics).

Writing in 1903, Ellis states³ that he had noted the exemption of the Europeans and Eurasians in 1890, and it has certainly continued ever since.

In the lunatic asylum Eurasians are dieted precisely like Europeans—that is to say, they are not fed on rice. The Javanese and Chinese attendants are rice-eaters, and get rations of the same rice as the patients. The Bengalis, keeping to their native habit, do not eat rice as a staple, and only occasionally as an adjunct.

21. In the **Singapore Prison** some 3,000 to 4,000 inmates have been received annually for the last thirty-five years. They are of mixed nationalities, and among them is a number of Europeans, which, though not in large proportion relatively to the total, is yet, absolutely, considerable. About 300 pass through the gaol every year, of whom about 10 per cent. are long-sentence prisoners, while the daily average number resident is about fifty.

In addition to the prisoners, some fifty or sixty warders are on duty constantly night or day at this gaol, about half of whom are Europeans, half 'Bengalis' (Sikhs and Pathans).

While the staple of all the native convicts in the prison is rice, that of the Europeans (and Eurasians) is bread. The Bengali warders also are non-rice-eaters, their staple being wheat-flour, made up with *ghee* into '*chupatties*' (a sort of cake of unleavened bread), which they prepare and cook themselves.

The native prisoners, classed according to grade, are housed

¹ *Ann. Med. Rep. S.S.*, 1898, p. 8, para. 33.

² *Ibid.*, 1899, p. 17.

³ *Ibid.*, p. 10.

in separate blocks, formerly many persons together in large 'association wards,' but at the present day each in a separate cell. The Europeans occupy one such building by themselves. The disposition of these blocks, five within a space of some 300 feet by 150 feet, make it incredible that any external agent of disease or means of infection which could have attacked one should not have affected the others.

Both through their contiguity, and the facts that the same warders move freely among all the prisoners, native and European, and that natives are often employed at work in the European quarters, there is every facility offered to the spread of any contagion from either class to the other.

There is thus a sufficiently large proportion of individuals not fed on rice, whose liability to beri-beri can be compared with that of rice-eaters, dwelling side by side with them, in one and the same place, and under conditions otherwise identical for all.

Beri-beri prevailed in the gaol almost continuously for the first fifteen years after its opening (1869-1884), becoming very severe in 1875-1876 and 1878-1881. After an interval of twelve years' freedom (1885-1897) it again appeared, in the autumn of the latter year, and scourged the prison until 1903.

The epidemic of 1878-1881 was carefully studied by Rowell, the last outbreak by Leask in 1898. The following data, compiled from the reports to Government of these two observers, show how severe the disease was :

Prevalence of Beri-beri in Singapore Prison.

	1878.	1879.	1880.	1881.	1898.	1899.	1900.	1901.	1902.	1903.
Average daily strength of } prisoners (natives) ... }	923	826	789	688	801	772	841	868	868	842
Number of cases of beri- } beri }	142	316	588	229	124	165	224	219	415	169
Incidence per 1,000 of } daily strength }	153	388	748	332	154	213	266	252	478	200

Throughout its earlier years, and especially during these two epidemics, therefore, this gaol may be regarded as having been a centre or hot-bed of beri-beri scarcely inferior to the lunatic asylum.

Under such circumstances, it is hardly conceivable that individuals of any nationality who were sufficiently long interned should escape.

Yet, as a fact, all the Europeans, all the Eurasians, all the

Bengalis, inmates or attendants of this gaol—in short, all those who were not rice-eaters—did so escape, and escaped alone, since the disease afflicted every other nationality.

There were, between 1869 and 1885, some 4,000 Europeans admitted, some 400 or 500 of whom served more than six months in this prison. Yet throughout that period, while the disease caused mortality among natives of all kinds at a rate seldom less than 24 per 1,000 per annum, Rowell states,¹ no European was ever attacked.

In the 1878-1884 epidemic the proportion² of natives and of Europeans attacked was respectively :

	1878.	1879.	1880.
Natives	4'1 per cent.	11'4 per cent.	20'7 per cent.
Europeans	Nil	Nil	Nil

The incidence upon the natives varied greatly, according to term of sentence, and with (though not on account of) nationality. Detailed figures appear in the table on p. 121.

The extraordinary incidence of the disease upon the Malays and Klings, as compared with Chinese, in this epidemic, out of proportion to their relative number in gaol at the time, and contrasting forcibly with the reverse state of affairs obtaining always outside, and even in the same prison in later years, affords an additional illustration of the importance of *time* in the production of beri-beri. For it will be seen that there was a very much larger proportion of long sentences among the Malays and the Klings than among the Chinese, the exact ratios being as under :

Percentage of Long Sentences among all Committals.

	1878.	1879.	1880.
Chinese	17 per cent.	18 per cent.	16 per cent.
Malays	39 "	44 "	29 "
Klings	29 "	37 "	33 "

This difference, it would seem, should alone be sufficient to account for the greater incidence of the disease on these two nationalities; but it is likely that a slight difference in the diet, the Chinese getting one meal of fat pork weekly where the Malays had only fish, helped to the same end. Possibly the Malays, who

¹ *Ann. Med. Rep. S.S.*, 1885; see *S.S. Blue Book*, 1886, ch. 30.

² The proportion taken is that of all cases of beri-beri occurring to all individuals passing through the prison during the year.

Incidence of Beri-beri on Different Nationalities in Singapore Prison.

	1878.			1879.			1890.		
	Long Sentences.	Short Sentences.	Total.	Long Sentences.	Short Sentences.	Total.	Long Sentences.	Short Sentences.	Total.
<i>Europeans:</i>									
Number incarcerated ...	24	246	270	29	266	295	25	180	205
Cases of beri-beri ...	}	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
Proportion per thousand ...									
<i>Chinese:</i>									
Number incarcerated ...	493	2,446	2,939	499	1,846	2,345	395	1,998	2,393
Cases of beri-beri ...	15	6	21	181	38	219			473
Proportion per thousand ...	30	2	7	362	20	92			19
<i>Malays:</i>									
Number incarcerated ...	105	164	269	103	128	231	72	175	247
Cases of beri-beri ...	75	7	82	61	3	64			82
Proportion per thousand ...	720	42	306	594	23	277			832
<i>Indians:</i>									
Number incarcerated ...	78	187	265	74	125	199	48	159	207
Cases of beri-beri ...	35	4	39	29	4	33			33
Proportion per thousand ...	448	21	147	392	32	166			169
<i>Total Natives:</i>									
Number incarcerated ...	700	3,043	3,743	705	2,365	3,070	515	2,332	2,847
Cases of beri-beri ...	125	17	142	271	45	316			588
Proportion per thousand ...	185	6	41	401	21	114			507

outside the gaols enjoy more generous food than the Chinese, may succumb more quickly than they to the effects of evil dietary, while neither Malay nor Kling is accustomed to uncured rice, such as was given in the gaol.

The same entire exemption of Europeans from the malady in this gaol was evident in the next and last epidemy. During all the six years 1897-1903, throughout which beri-beri lay heavily upon the native convicts, *not a single European prisoner was attacked, nor any one of the European or Bengali warders.*

The nationality of the cases occurring in the last epidemy was as follows :

Distribution of Cases of Beri-beri according to Nationality in the Singapore Gaol, 1898-1901.

	1898.	1899.	1900.	1901.
Chinese	116	158	203	214
Malays	5	5	16	1
Indians	3	2	5	4
Total native cases ...	124	165	224	219
Europeans	<i>Nil</i>	<i>Nil</i>	<i>Nil</i>	<i>Nil</i>

That nothing in their location existed to protect the European convicts is a point made salient by a remark of the surgeon in charge in 1900, Dr. J. Kirk, who says : ' Her Majesty's Pleasure Karim (a Malay), who worked and slept in the European block, contracted beri-beri and died. There was, however, no conveyance of infection to any of the European prisoners.'

The exemption especially of the Europeans is further demonstrated by the following figures, showing nationality of all cases of sickness treated in the prison hospital from 1899 to 1901, for which I am indebted to the courtesy of the late Principal Civil Medical Officer for the Straits Settlements, Dr. W. S. Kerr :

Analysis of Cases treated in the Singapore Prison Hospital, 1899-1901.

	Admissions for all Diseases.	Admissions for Beri-beri.	Proportion of Cases of Beri-beri to all Diseases.
Europeans	51	Nil	Nil
Chinese	2,609	575	19 per cent.
Malays and other natives of the Archipelago ...	236	21	9 "
Tamils	223	10	4 "
Other natives of India ...	13	2	15 "

A priori, it must seem that permanent attendants should be, from mere length of exposure to the possible exciting cause of beri-beri, more liable to acquire it, in an institution where it is prevalent, than are their charges, who reside in the focus of its occurrence for much shorter periods; and the experience of the asylum shows that a considerable proportion of the attendants there did acquire beri-beri. These were rice-eaters.

But at the Singapore Prison the warders, mostly Bengalis and Europeans, have never been attacked, a singularity of divergence which is to be explained only by the fact that they are not rice-eaters.

Eurasians in prison have the same diet as Europeans. The exemption of Eurasians in asylum or prison is the more noteworthy, since outside these institutions the habits of the class are those rather of the native.

They are almost all rice-eaters, using indiscriminately every sort of rice. Accordingly, they are, when free, considerably subject to beri-beri.

From the returns given in a later table (see p. 194) it will be seen that of 387 'Europeans and Eurasians' admitted to the Singapore Pauper Hospital from 1899 to 1901 (nearly all of whom were Eurasians, *not* Europeans), six Eurasians had beri-beri. In the Penang General Hospital during the same years, of 591 admissions of Europeans and Eurasians, mostly the latter, five Eurasians had beri-beri.

22. Stanley, summing up his conclusions from a study of beri-beri in the **Shanghai gaols** (four in number), says 'the incidence in four widely-separated prisons, completely isolated in every respect, was of approximately the same degree. *In none of these places are the European and Indian staff affected*, although residing in the same compound with the prisoners. The cause of the disease does not arise, therefore, either in the soil or their immediate surroundings.'

I shall content myself with insistence upon the one clear fact manifested in these prison epidemics, as in that at the asylum—that in a mixed community of persons, all equally susceptible, and all equally exposed, in a really severe focus of beri-beri, continuing year after year, *those only who were rice-eaters were attacked; those who were not rice-eaters, whether European or native, whether warders or convicts, escaped.*

The above are all instances of small communities of persons,

initially healthy, suddenly completely isolated from the rest of the world, so that indeterminable factors (of intercourse, infection) were eliminated, placed in each case in an environment the same for every individual—in the case of the prison, air, water, soil, drainage, housing, clothing, bedding, washing, work, exercise, were identical for all.

Every individual was susceptible to beri-beri, none had or could have gained immunity through previous attacks. The only difference between members of these groups was in their food, and in each instance, when the disease appeared, there was a definite selection of the patients, and the incidence was always upon those who did, never upon those who did not eat rice.

Evidence from Difference of Diet due to Ethnological Characteristics.—It is not only in confined communities, in restricted localities, or particular sorts of institutions, that the immunity of non-rice-eaters is witnessed. It is apparent wherever, in a region in which beri-beri is endemic, races differing in this habit live side by side.

In the Straits Settlements and the native Malay States of the adjacent peninsula, the conditions of which will be presently more particularly referred to, alone among all the races occupying the territories, the (British) Europeans and tribesmen of North and North-Western India (Sikhs, Pathans, Panjabis, Bengalis) are not rice-eaters, and they alone¹ of all the nationalities escape beri-beri.

In speaking of 'Europeans' in this connection, British, Germans, and others born in Europe, Americans, and British subjects of colonial birth alone are to be understood as referred to, since the native-born Europeans, chiefly Portuguese and Dutch, differ from them in the habit of food, being, like Eurasians, almost all persons whose staple of diet is rice.

The quantity of this grain consumed by those whose principal article of food it is is not small. A pound to a pound and a half of the dry rice forms an amount commonly consumed by an individual in a day. But in the diet of those whom I have classed as Europeans, on the other hand, even when, for variety's sake, a small quantity of it is added to their meals, rice never forms a component of any importance. The diet of the European sojourning in British Malaya, at least, is always as ample and varied, and as nearly similar to that to which he is habituated in Europe as it is possible to make it.

¹ I exclude from consideration here the Tamils, who, although rice-eaters, are also—so long as they eat only a special sort of rice—exempt from the disease.

If the Europeans in Malaya are non-rice-eaters, the Central and Northern Indian natives, immigrant into the same region, are no less strictly in the same category. In their own country their staple of food is millet or wheat-flour. This is made into a paste with *ghee* or other fat or oil, but no leaven, rolled out into a sort of pancake (*chupatti*), and cooked on a hot iron or stone. No race is so meticulously observant of the prescriptions of caste in the preparation of food as are Sikhs and Hindus, for whom it is a contamination if even the shadow of an unbeliever fall upon their food during its preparation. Mohammedans are little less so, and it is to one or another of these three divisions of belief that nearly all the Indians here classed as 'Bengalis,' including Sikhs, Pathans, Panjabis, and other natives of Bengal proper immigrant into Malaya, belong.

When to religious prejudices are wedded also the influences of social custom and physiological habit, it is not surprising to find that the Bengali, whatever the country to which he migrates, adheres with rigorous persistence to that rule of life in all its details, and to the exact kind of diet to which he has been accustomed from his infancy.

In his own country he never eats rice, and although he may, like the European, add it occasionally as a variant to his dietary, the ordinary Sikh or Bengali never becomes solely a rice-eater in any land of his adoption. While this refers only to, and is generally true of, all those who are comparatively well-to-do and in respectable employ, as the military police (the bulk of whom these natives here compose), indigence, economic reasons, and the tendency to become assimilated to their surroundings bring it about that exceptions occur, and some few among the Bengalis do adopt rice largely for food. But even among such cases it is seldom made the staple diet. As a class they are non-rice-eaters.

In 1891 there were in the Straits Settlements and the adjoining native States of the peninsula 5,365 Europeans; in 1901, 3,696, the decrease being partly due to the withdrawal of a military garrison. The Bengali element numbered in the same years 5,991 and 8,507 individuals.

Male adults only are counted, for the purpose of comparison with other races, since the females and children of the native classes (except Tamils) do not come to the hospitals.

This number of persons must be conceded as affording an amount of material quite sufficient for the demonstration of the

degree to which either of these two races should be liable to get beri-beri when placed in the circumstances in which the disease is acquired by others.

A later table shows the distribution of the two classes geographically in 1901 (p. 134).

As regards circumstances of life, it is to be remarked that the non-military Europeans in the urban settlements are principally traders, and in the native States chiefly civilians in Government employ. Their circumstances might be expected to afford them some degree of exemption from factors determinative of beri-beri, were those factors of a kind naturally connected with the habits or occupations of the lower or coolie classes (whom the disease chiefly affects), such as filth, indigence, overcrowding, overwork, exposure. But many are planters, miners, prospectors, and contractors, the necessity of whose avocations exposes them often to equal fatigues, and always to contact with natives and so to opportunity of infection. The isolated cases of beri-beri which do occur in Europeans, moreover, show that such differences in circumstances of life do not avail when the real causal factor (which is none of these) affects them.

For the Bengalis, most of them are, as has been said, military police. Housed in barracks, they are so far in a condition which has been supposed, if fallaciously, peculiarly to favour the spread of beri-beri. Of the remainder, a large number are employed as watchmen in shops and mines.

At every important mine, and even at many small ones, it is customary to employ a Sikh or Bengali watchman, who is armed, for the protection of the headmen against possible rioters, and of the mine's property from thieves, etc. Many of these watchmen are employed, and remain for long periods, living at exactly those centres, and even in the very buildings, in which beri-beri is rife among the coolies.

Under these circumstances it is difficult to conceive any means by which they should be able to preserve themselves from any cause of disease due to their surroundings which would be operative upon their fellows. Were they found to be—and this is the case—constantly so preserved, therefore, the fact would be one of no little significance, and demanding explanation.

Other Bengalis engage in trade as cattle-keepers and bullock-cart drivers. To the latter especially as a class attach, more than to any other section of the coolie population, precisely those deteriorating influences of dirtiness of person and habitation,

and of penuriousness of life, which are supposably connected with the spread of most diseases. Were it possible for dirt alone to determine beri-beri, the low-class Bengali would surely never escape it.

It may be added that of all sections of the community the Bengali is the most drunken, and the influence of alcohol predisposes, my own knowledge of certain European patients leads me to believe, to beri-beri, as it does to other forms of peripheral neuritis.

In the Europeans and Bengalis we have, therefore, a population of some 10,000 individuals, occupying for a period of ten years (or more) an area throughout which beri-beri has been constantly endemic and epidemic, and whom nothing in their outward circumstances prevents the disease from attacking. There is, it is agreed on all hands, no such thing as a racial immunity from beri-beri. Unless, therefore, the Europeans and Bengalis should have some therapeutic means of protecting themselves from the disease, or something in their habits should save them from its incidence, it is incredible that they should throughout this area, during this period of time, almost uniformly escape the disorder. What, then, is the fact? Do these two races escape beri-beri?

The answer to this question is definite, and may be to some astonishing. The fact, broadly stated—so far as I have been able, I have assured myself of its accuracy—is that, so long as he keeps to the habits of his class, *so long as he does not eat rice, no European, no Bengali, in this region ever acquires beri-beri.* Throughout the area, throughout the period dealt with, this population of 10,000 Europeans and Bengalis has been, and is, absolutely exempt from beri-beri.

23. Immunity of Europeans in British Malaya.—The writer, during fourteen years' medical charge in the Malay Peninsula, has personally met with but three, and has been able to become acquainted with the circumstances of but four more, cases of beri-beri in Europeans. Each one of these patients, from choice in four, and from necessity—the conditions entailed by long jungle excursions—in the three others, was a rice-eater—*i.e.*, one of whose diet rice was always or became for the time the chief constituent.

The limits of a single personal experience in this particular are fortunately extended by the ampler records of hospitals in the Straits Settlements.

The Singapore General Hospital, in which patients of every

nationality are received, admits some 500 Europeans annually. Were cases of a disorder so incapacitating as beri-beri to occur among the Europeans, it is inevitable that some at least of them should find their way into this hospital.

The European adult male population of Singapore numbered in 1901, when a census was taken, only 2,273. In that year 438 patients were admitted to the wards, a ratio of 192 per 1,000. The admissions for Chinese, who numbered 118,196 adult males, to all hospitals at the same station for the same year was 8,325, a ratio of only 63 per 1,000. So that the proportion of sick Europeans treated in hospital was more than thrice as great as the number of Chinese, an excess of sickness rate which is only partly accounted for by the fact that this hospital receives patients from ships touching at the port.

Now, among the Chinese, of all the cases treated, no less than 139 out of every 1,000 had beri-beri.

But *among the Europeans there was not a case.*

The returns for the two previous years reveal similar results. In all three years 1,530 Europeans were treated at the Singapore General Hospital, not one of whom had beri-beri.

In the Penang General Hospital also a considerable number of Europeans are treated. In the returns which I have obtained for this hospital, however, the distinction between Europeans and Eurasians—an all-important one when diet is in question, Eurasians being many of them rice-eaters—has not been made. The greater number of the cases included in the Penang return were Eurasians.

At Penang, among 387 cases from both classes treated in three years at the General Hospital, but *five* were cases of beri-beri. Two of the patients were 'Government subordinates from Perak,' a description which implies that they were, if not Eurasians, at all events persons in a station of life in which rice would almost certainly be the staple diet. Two were Eurasians. The fifth was one of the three cases of beri-beri in Europeans mentioned by the writer as having been seen by himself—in fact, a patient who had been under his own care—and he was certainly a rice-eater. The Penang General Hospital yields, therefore, the same evidence as that at Singapore. No Europeans of *European habits of life* were treated there for beri-beri.

At the Singapore Pauper Hospital 591 'European and Eurasian' patients were treated in three years. Of these seven had beri-beri, one of whom only was European, a Scotsman. At

Penang Pauper Hospital for the same period 148 Europeans and Eurasians were treated, two of whom had beri-beri. Nearly all the cases included in the group mentioned at both these pauper hospitals were naturally Eurasians. It is hardly necessary to point out that the presence of a European among natives in a pauper hospital indicates destitution, and the probable adoption by the patient of the habits of the class to which he has become reduced.

The considerable number of *Eurasian* cases of beri-beri is to be taken note of as showing the greater incidence of the disease upon this, a partly rice-eating class, than upon others which are not so.

In effective contrast with the experience of the Dutch in their neighbouring possessions, the statistics of the British Malayan Settlements show, therefore, that the European, even in an area in which beri-beri is endemic, does not acquire it so long as he retains the food habits of the European.

24. Immunity of Bengalis in British Malaya.—Still more significant is the exemption of the Bengali class. Although large numbers of these men are housed in barracks; although many are of low class, dirty and penurious; although, as has been stated, many of them are employed at exactly those centres, live even in the very habitations, in which the disease may be most pestilentially prevalent among the Chinese coolies, yet it is a fact that never does one of them, so long as he preserves the dietetic habit of his class, become attacked with beri-beri.

I have but twice seen beri-beri in Sikhs,¹ only once in a Bengali proper. The last is a patient—a bullock-cart driver—under observation at the time of writing, suffering from a mild attack, who is, upon his own statement, a large eater of rice, and has long been so. Although he prefers *chupatties*, he says he also likes rice, and, finding it cheaper, makes it his chief article of food. At the Seremban Hospital, under the writer's charge since 1891, 377 Bengalis were treated during ten consecutive years. Not one had beri-beri.

It is not that Bengalis, as a class, are generally less susceptible to disease than other classes. Most of the patients in this

¹ These two cases occurred before I had gained my present conviction as to the connection between rice and beri-beri; hence I did not inquire what *kind of rice, if any*, they were in the habit of eating. But they were bullock-cart drivers, a class more commonly adoptive of local habits than others.

hospital came from the small force of military police kept at Seremban, which averaged sixty-four in strength, with 63 per cent. of admissions to hospital, annually during the ten years. Nor is it that they are secure either from any presumable outward cause of infection, if there be one, which can produce beri-beri, or even from actual contact with persons suffering from the disorder. At Seremban the barracks of the Sikhs are closely adjacent to those of the Malay police, and the two classes are constantly in close communication; yet the Malays, at the same time and place, although furnishing a much smaller number of admissions to hospital, are considerably affected by beri-beri. During the same decade, of 102 admissions of Malay police to hospital, 10 were for beri-beri; and this represents less than the true proportion, since the sick Malay almost always goes to his own home.

In the Straits Settlements the experience is the same, although I have been unable to secure returns which make this point as clear as it might be, since in those with which the kindness of my colleagues has provided me the distinction between the different classes of Indians (other than Tamils) treated has not been made. Sikhs, Pathans, Bengalis proper, Malabarese, and even Sinhalese are in the tables on pp. 133 and 196, included together under the heading of 'Other Natives of India.'

Nor was information obtainable at the time when the return was prepared as to what might have been the departures from their own customs in regard to rice-eating of any of the patients treated for beri-beri, who were actually included in this class. Nevertheless, admitting all these sources of error (the effect of which was solely to magnify the incidence upon the Bengalis), among 2,220 'Bengali' patients treated in the hospitals of the Straits Settlements during the years 1899-1901, only 21 had beri-beri, four of whom were Sinhalese,¹ one a Malabarese, one a Panjabi pauper, the rest 'Bengalis.' Of the latter, five were paupers. Accepting the sixteen Bengalis (with the Panjabi), the return shows that, of every 1,000 cases of disease admitted to hospital from among the Bengali class, only five were beri-berics, as to none of whom it could be affirmed that they were not, while the probabilities are that they were, rice-eaters. With this may be compared the rate obtaining at the same time and place among Chinese, all rice-eaters, which

¹ Rice-eaters.

was 107 per *mille*. A contrast, this, the import of which is heightened by the reflection that probably every case of serious disorder among the Bengalis finds its way into hospital, the bulk of them being police under medical supervision, and all as a class being well acquainted with, and confiding in, European ways and treatment, so that instances of a disorder like beri-beri could hardly escape notice; while among the Chinese enormous numbers are affected by, and even die of it, who for precisely opposite reasons never come to account.

At Penang 1,249 Bengalis, chiefly Sikhs, were treated, without a single case of beri-beri.

Among 4,327 Bengali patients, all that were treated at the hospitals, in the years for which I have been favoured with statistics, there were altogether twenty-three cases of beri-beri—5 in 1,000—every one of whom, the probabilities are, ate uncured rice habitually.

The criticism that might be addressed to conclusions which are based upon small numbers, or which might be affected by the variability of the incidence of the disease over restricted areas, or for brief periods of time, cannot apply here. If during the ten¹ years in which the whole of the native Malay States as well as the Settlements have been under close medical supervision—a period so long as to exclude possible seasonal, or even multiannual mutations—a population so considerable as the Europeans and Bengalis compose, scattered over an area so large, have remained uniformly free from a disease to which their neighbours of every other nationality at the same time and place succumb, the source of their protection must lie in something common to the two races which are exempt, something not shared by the races which are attacked. There is no extrinsic factor which will meet this requirement. It must obviously be some personal habit or national custom, capable of determining disease-incidence, shared by all the members of each race.

Now, between Bengalis and Europeans there is no single habit of this nature in common other than the fact that neither are rice-eaters. On the other hand, between these two races which are free from beri-beri, and those—Eurasians, Chinese, Malays,

¹ Or fifty, for the results have been the same, although they are not tabulated ever since the Settlements have been founded, or the native States opened.

Javanese, Boyanese, and even Sinhalese—who are not so, this habit alone marks a constant difference. All the latter do eat rice.

Sufficient has been said now, it may be conceded, to prove that *so long as they preserve their own national habits, so long as they do not eat rice, Europeans and Bengalis are, even when living in severe foci of beri-beri, completely exempt from it.*

This immunity disappears when rice becomes the chief article of their food.

The converse of the statement made above then becomes, so far as evidence on the point is obtainable, equally true. *When they do eat rice of an uncured sort for any length of time, both Europeans and Bengalis get beri-beri.*

In regard to Europeans, the cases mentioned by the writer as having come under his own personal observation are convincing examples. In none of those three cases was there any unusual factor likely to be a special cause of disease present, except the habitual and excessive use of uncured rice in their diet.

The prevalence of the disease among the Dutch troops in Sumatra, in their navy, and, indeed, among Dutch colonists generally, which is a matter of notoriety, affords a body of evidence to the same end ampler in its extent.

In regard to Bengalis, less evidence of the effect of reversed conditions of diet in producing beri-beri is obtainable, but so far as it goes it is in accord with the theory.

I have mentioned that the only Bengali I have myself seen with beri-beri, in whom this point was ascertained, was an eater of uncured rice.

In the Singapore Prison, although the number of Bengalis admitted is absolutely not large (some thirty to fifty annually), and although still fewer of them are incarcerated for periods long enough to acquire beri-beri, one case occurred (in 1901) among thirteen admissions of Bengalis to the infirmary for all causes during the three years 1899 to 1901.

In this gaol all prisoners get a certain allowance of wheat-flour in addition to their rice, *but the staple of the diet is¹ rice, for Bengalis equally with the other nationalities.*

In the Pudooh Gaol in Selangor, in which beri-beri has been severely prevalent since August, 1895, Bengalis receive the same

¹ Or was.

134 THE CAUSE AND PREVENTION OF BERI-BERI

diet of uncured rice as the other prisoners.¹ Although very few of the nationality are incarcerated altogether, two Pathans acquired beri-beri there during the eight months of an observation carried on by H. Wright ('Study,' par. 340, p. 52).

The tables on pp. 133 and 134 show the distribution of Bengalis and Europeans geographically in British Malaya in 1901, their relative general rate of sickness, and the proportion of beri-beri to other diseases.

Table showing Population, Admissions to Hospitals for all Diseases, and Admissions for Beri-beri, for Three Races, in 1901.

DISTRICT.	POPULATION. MALE ADULTS ONLY.			ADMISSIONS TO HOSPITALS.					
				For all Diseases.			For Beri-beri.		
	Chinese.	Euro- peans.	Ben- galis.	Chinese.	Euro- peans.	Ben- galis.	Chinese.	Euro- peans.	Ben- galis.
Singapore ...	118,196	2,273	2,500	7,339	705	507	1,102	1	9
Penang ...	44,563	488	813	3,763	229	554	447	1	3
Province Wel- lesley ...	20,732	99	441	620	—	57	28	—	—
Malacca ...	13,179	48	53	2,653	1	39	240	—	—
Total Straits Settlements	196,670	2,908	3,807	14,375	935	1,157	1,817	2	14
Perak ...	132,553	383	3,244	17,017	87	924	4,760	—	—
Selangor ...	94,297	306	1,086	10,862	?	?	2,065	?	?
Negri Sembilan	30,762	99	370	1,886	—	167	1,104	—	—
Pahang ...	8,093	103	281	884	4	292	230	—	—
Total Native States ...	265,705	891	4,981	30,647	—	—	8,159	Nil	Nil

The first and most important among all the deductions from the rice theory of beri-beri has thus been completely proved. Wherever facts have been available for evidence—and this is the case in regard to an immense number of persons, under widely

¹ This applies to 1901 and previously. By a subsequent alteration of diet Bengalis have become entitled to draw, if they prefer it, wheat-flour instead of rice.

Table showing Proportions of Population Admitted to Hospitals for all Causes, and Proportions of these which had Beri-beri, per 1,000 for same Year and Places.

DISTRICT.	CHINESE.		EUROPEANS.		BENGALIS.	
	Ratio per 1,000 of Admissions to Population.	Ratio per 1,000 of Beri-beri to all Admissions.	Ratio per 1,000 of Admissions to Population.	Ratio per 1,000 of Beri-beri to all Admissions.	Ratio per 1,000 of Admissions to Population.	Ratio per 1,000 of Beri-beri to all Admissions.
Singapore ...	62	150	310	1.4	202	15.9
Penang ...	84	118	496	4.3	67	5.4
Province Wel-						
lesley ...	40	45	Nil	Nil	129	Nil
Malacca ...	201	90	Nil	Nil	735	Nil
Perak ...	120	279	—	—	—	—
Selangor ...	115	190	—	—	—	—
Negri Sembilan	61	585	Nil	Nil	451	Nil
Pahang ...	109	230	39	Nil	1,039	Nil

different circumstances—they show that in mixed communities, *wherever beri-beri is endemic, or epidemic, its incidence is solely upon those who do, never upon those who do not, eat rice.*

SECTION IV (*continued*)

PROOF OF DEDUCTIONS MADE FROM THEORY OF GRAIN INTOXICATION IN CASE OF RICE

II

AMONG RICE-EATERS DURING EPIDEMIES, AND IN PLACES WHERE
BERI-BERI IS ENDEMIC, ITS INCIDENCE VARIES WITH THE
SORT OF RICE EATEN.

Only Certain Sorts of Rice Toxic.—Both the extent, the frequency of occurrence of cases in a community (the case-incidence), and the severity of the disease (the case-mortality) should vary with the sort of rice customarily eaten.

Considering the enormous number of varieties of rice grown,¹ the poverty of our knowledge of the parasites and saprophytes of the grain, the absence of direct evidence that a definite poison is extractable from any of them, and recognising the impracticability of tracing the source and subsequent distribution of small parcels of the material, the difficulties of establishing a connection between the prevalence of beri-beri and the consumption of particular sorts of rice might seem wellnigh insuperable.

But it is this very evidence, the absence of which Manson² declared made it impossible to accept any theory of the dependence of beri-beri upon rice, that the facts I am about to detail will supply. It is evidence which could not have been obtained by those who have studied beri-beri previously, because the conditions which have produced it have themselves only recently been called into being.

¹ 'At the Calcutta Exhibition of 1884,' a writer in 'Chambers's Encyclopædia' states, '4,000 apparently distinct forms of *Bengal* rice were shown. There are 1,400 different specimens of rice in the Calcutta Museum' (ed. 1891, art. 'Rice,' vol. viii., p. 702).

² Davidson, 'Hyg. and Dis. Warm Climates,' p. 490.

Although, then, the botanic varieties of the cereal producing rice are multitudinous, the forms in which it appears commercially are few and easily distinguished. Broad differences are established by divergence in the processes followed for separating the rice from the raw grain, both as to appearance, as to food-value, and as to toxic potentialities.

In this section the character of the chief rices in consumption will be described, the relations of beri-beri to their consumers traced, and the clear connection of the disease with the consumption of particular sorts of rice will be shown.

The influences which may render such different rices unequally toxic will be discussed later, when their causal relation to the malady has been fully established.

Structure and Composition of Rice-Grains.—To appreciate fully the extent to which the character of rice may be affected by different processes of dressing—husking, milling, and preparation for table—some reference must first be made to the structure and composition of the individual rice-grains.

The raw grain, *padi*, the ripe fruit of the cereal grass *Oryza sativa*, like that of other cereals, is described botanically as a single-seeded, dry, enclosed caryopsis. It consists of an ovule (ovary, endocarp, endosperm) invested by layers successively designated as the episperm (testa), mesocarp, and epicarp (with a reliquary surface epidermis). The two latter, mesocarp and epicarp, together form the pericarp proper, and the whole three the 'pericarp' commonly so called, or 'inner skin,' or 'zilver-vliesjes' of the Dutch. Finally, the two closely-united persistent inner glumes or paleæ, one slightly overlapping the other, form the adherent indehiscent outer shell or husk. The husks, like the pericarp, vary in colour, according to variety, the range being from black to light yellow; the commonest is buff or barley-colour.

The husk is a comparatively thick-ribbed mass of withered woody cells and fibres, for the most part arranged longitudinally. Wet or dry, it is when ripe very brittle and easily split, and requires no further description.

The parenchyma of the ovary (or the endosperm) is composed of large, irregularly oblong cells, stuffed with starch granules of the shape peculiar to the grain—i.e., of rounded angular shape—five to six, or sometimes three sided, of a size smaller than the small granules in wheat, about $\cdot 005$ mm. in diameter,¹

¹ $0\cdot 005$ to $0\cdot 0076$ according to Blyth; $2\ \mu$ to $10\ \mu$ according to Macé ('Les Substances alimentaires,' etc., 1891, p. 277).

mostly of uniform size, and with an inconspicuous outline, sometimes packed together in larger ovals, and sometimes the whole contents of one cell.

The surface of the ovary is occupied by a layer of cells, usually single, but sometimes double, consisting of cubical cells, elongated axially, in which the substance of the seed is stored as gluten. This layer is readily separable from the rest of the pericarp, and is then also firmly adherent to the pericarp, and is separated with the latter when the grains are threshed.

The proteid or aleurone grains appear as small, round granules, much smaller than the starch grains, yellowish-brown with iodine, red with cochineal, and with water turn into oily-looking drops. The fat usually found in rice is associated with the aleurone, and is found partly in the surface layer, partly in the aleurone.

The pericarp, as already mentioned, is in some sorts coloured some shade of red. A deep brick red is the colour of the grain commonly grown in the East, but there is much grain in which this layer is absent, in some sorts it is yellow, brown, or even black.

The pericarp has square cell layers of varying thickness, withered, coloured, or sometimes colourless cells (the mesocarp), upon which lies an epidermis of polygonal cells, difficult of detection (all that there is of it is lined by a simple utricle of slender intercellular spaces, the episperm). It varies in thickness and colour in different sorts, as well as in the tenacity of its adherence to the ovary.

Its woody consistency makes it, too, fairly brittle, and it fractures, even after prolonged soaking in water, into small pieces.

The pericarp firmly adheres on the one side to the ovary, on the other to the husk, by simple apposition, all over, so that there is no structural union of the three. The seed furrows, is lightly 'ribbed,' but there is no deep furrow, as occurs in wheat, barley, etc., and there are no ribs, so that the pericarp into it, so that there is no obstacle to its removal in milling.

The extent of adhesion of the pericarp to husk or ovary on the other, varies in different sorts, and is of importance, owing to the superficial gluten-cell layer. Thus Vorderman¹ says in J

¹ 'Onderzoek,' etc., p. 11.

exposed for sale rice which has all its pericarp still attached and rice without any, both of which sorts have, however, been submitted to the same process of milling, had the same amount of labour spent upon them, and have been grown in the same district. The 'red rice' (as the grains with adherent pericarp are called) retaining all the gluten, is necessarily more nutritious; and, what may be of even greater significance, the grains have in this still adherent investment a protection against penetration by damp, by moulds, and other saprophytes.

Practically the whole of the albuminous matter of rice and most of the fat is stored in the gluten, which is found only in the surface, the aleurone-layer of cells. The proportion of gluten in rice is given by most authorities as about 7 per cent., compared with an average of 12 per cent. in maize, 14 per cent. in oats, and 22 per cent. in the best wheat.

The following analyses of rice of various samples, with some of wheat and maize for comparison, show its food value when whole (with pericarp) and when stripped :

No.	SAMPLE.	COMPOSITION.					
		Proteid.	Fat.	Carbo-hydrate.	Cellulose.	Water.	Ash or Salts.
1	Wheat-flour	11.0	1.0	74.9	—	—	—
2	Corn-meal	9.20	3.8	70.6	—	—	—
3	Rice	7.4	0.4	79.4	—	—	—
4	Wheat	12.4	1.4	67.9	1.8	13.6	1.8
5	Rice	7.9	0.9	76.5	0.6	13.1	1.0
6	Rice, cleaned white	2.0369	0.5068	82.6485	0.5757	13.736	0.4961
7	White rice	8.50	0.51	74.11	0.66	15.67	0.55
8	Red rice	10.34	1.38	71.04	1.52	15.06	0.66
9	Stripped B.I. rice	7.3	0.6	78.3	0.4	12.8	0.6
10	Fine rice offal, ¹ best sort	13.6	14.7	44.0	8.0	10.7	9.0

The analyses given are : Nos. 1, 2, and 3 by Woodruff, quoted by Thompson ;² Nos. 4 and 5 by Munk, from Halliburton ;³ the remainder, No. 6 by Maier, Nos. 7 and 8 by Scharlee and Bernelot Moens, No. 9 by Church,⁴ No. 10 by König, are taken from Vorderman.⁵

¹ *Dedek lunti*, or *dedek halus*, the offal from the last grinding = sharps.

² W. G. Thompson, 'Practical Dietetics,' 1902.

³ 'Chem. and Phys. Path.,' p. 598.

⁴ 'Food-grains of India,' p. 73.

⁵ *Loc. cit.*, p. 25.

In few of them is the state of the sample, the extent to which it was decorticated, upon which its composition so obviously depends, even mentioned.

It is clear from the analyses, however, that, as already stated, most of the proteid becomes removed with the pericarp; so does the fat.

The extremely small proportion of *fat* in rice, even more than its comparative poverty in proteid, renders it inferior to other cereals as an article of diet. Taken alone, rice is incapable of supporting life, at least in health and activity, since, to obtain the amount of *proteid* reckoned as sufficient for barely sustaining life—viz., 75 grammes daily—somewhat more than a kilo of ordinary stripped white rice must be taken, while to furnish the necessary amount of fat—40 grammes—not less than 10 kilos of the same rice would be requisite.

It is manifest from this that in rice, more than any other cereal, it is essential to preserve intact the proteid element.

The preservation of that proteid depends entirely upon the integrity of the cells containing the gluten, which form a thin layer on the surface of the seed.

¶ The manner in which rice is separated from the raw grain is therefore of great importance; for it is obvious that if care be not taken to preserve the gluten layer the dietetic value of the product, so far as its proteid equivalent is concerned, may be reduced almost to nil.

In a dry state the husk of rice is easily removed. Light tapping or pressure, the attrition of the grains against each other in a closed space or between rollers, causes it to crack and split off. Some of the pericarp, adherent to the husk, flakes or scales off at the same time, but completely to remove the latter requires special treatment, more pounding, or friction under greater pressure, and the two operations are generally made separate stages in the various processes of milling.

It is under these circumstances that the peculiar physical relation of the pericarp and the endosperm (or at least the latter's outer layer) becomes of importance. For, when the seed is dry, the adhesion of the pericarp to it is such that when the former is removed by crushing (pounding or friction), the surface layer of the ovary (the aleurone or gluten layer) cleaves to and is removed with it. Thus the more thoroughly it is decorticated the less valuable as food does the grain become.

Rice from which only the husk, and not the pericarp, has been

removed, commonly red, is called 'red' rice. When all the pericarp is cleaned off the product is 'clean' or 'white' rice.

Maceration of the grain, especially in warm water, causes the envelopes to become more distinct and more easily separated from each other. Neither husk nor pericarp is much softened, but the seed, absorbing water, gradually softens and swells.

If the grain be boiled, or heated slowly in water at about 80° C., the ovary, greatly enlarged through the swelling of its starch-grains, bursts the pericarp and husk, and at the same time complete separation of the various envelopes is effected, so that the paleæ become loosened from pericarp, and the latter from the aleurone layer of the seed. If then thoroughly dried, the ovary shrinks to its former size, the ruptured pericarp still adhering to it, the split husk forming a loose wrapper, which is now easily dislodged by simply rubbing with the fingers, or, in native practice, with the feet.

The pericarp after boiling, although burst in places, elsewhere remains still adherent to the seed, but is more easily removed by fracture (pounding) or by attrition (milling) than in seed not so treated.

But now the aleurone layer, instead of separating with the pericarp as it usually does in unboiled grain, remains firmly adherent to the ovary, the surface of which, owing to its presence, appears smoother than that of rice which has not been macerated before milling.

Methods of Preparation.—The methods of preparation of rice from padi actually practised are as follows :

I. *Fresh Rice.*—As grown by the Malays of the Peninsula for their own consumption, padi is reaped by cutting off the ripe ears one by one. The grain is dried in the sun, and garnered in bark bins, which are built under the house, or in as dry a place as possible. From this store a portion is taken daily, as required for food, and hulled by pounding it with a wooden pestle in a wooden mortar. The mortar is a deep hollow excavated in a log of hard wood, the pestle a thick, smooth peg inserted into the under side and at one end of a long beam. The latter works on a cross-pin fixed close to the end farthest from the pestle. Here the operator stands. A slight movement of the foot, depressing the short arm of the lever so arranged, tips up the long arm, carrying the pestle to a height of a foot or two, whence it falls into the mass of grain placed in the mortar beneath. A repetition of fairly heavy blows is thus delivered with little effort.

The grains coming under the pestle at each fall are tapped or bruised, but not broken, as they rest on the elastic mass of other grains below.

The rice, thus thoroughly husked (stripped, shelled, bolled, or hulled) is winnowed free of chaff, and returned to the mortar, to be completely decorticated or freed of pericarp by repetition of the previous process.

Winnowing is effected by placing grain in small quantities on shallow oval trays of plaited grass or reed, with raised edges, rolled over inwards; the mass is tossed upwards into the air; the more rapidly-descending grains, caught first on the tray, are freed from the slower-falling bran, which floats or is blown away.

According to the sort of rice, its size and condition, dryness, the toughness or adhesiveness of the envelopes (especially that of pericarp to ovary), the amount of labour required for husking and the time spent on it varies. Usually the hand process is effected in two or three stages and in mortars of different size, the larger being used first. At the first stage the stalks, coarse husks, etc., are removed, the offal being straw; at a second all the husks and some of the pericarp—offal coarse bran. A third stage removes fine offal, chiefly of pericarp, with some starch (portions of seed), leaving imperfectly white, but eatable, table rice. This stage is that in which it is commonly eaten in native households. Further pounding, etc., produces perfectly clean, wholly decorticated white grains. Such fine rice is eaten principally by the higher class Malays, households of native chiefs, etc., where there is plenty of labour to spare for its production.

The grain during these proceedings is frequently turned over by hand and narrowly inspected, so that dirt and discoloured or apparently bad grains are readily seen and removed.

Vorderman¹ gives the following rough analysis of an ordinary table rice as found in Chinese stores in Batavia :

Well-cleaned rice, whole grains	80
" " broken "	10
Coarse bran	5
Fine bran or sharps	5
					<hr/>
					100

Vorderman also states² that in fine-grained varieties the pericarp forms about 4 per cent. by weight of the whole grain (without husk). Of this 4 per cent., possibly one-tenth—4 parts in 1,000—might be gluten.

¹ 'Onderzoek,' etc., p. 14.

² *Loc. cit.*, p. 21.

Before cooking the rice is laved copiously with water. The object of this is chiefly to remove the dust and small particles of starch which would make the grains stick together when boiled, whereas the Malay prefers every grain in the cooked mass to remain quite separate. But in the process many diseased grains float and are rejected, heavier discoloured ones are seen and picked out, and the rice is thoroughly cleansed of all extraneous matters by the repeated rinsings.

If it seem that the proceedings detailed show an elaboration of care beyond the wont of Orientals, it is to be pointed out that the women, whose household care this is, have little else to do; the preparation of the meal is the chief labour, as its consumption is (in more places than the East) the principal pleasure of the day, and in the humble economy of the peasant's home the production of the chief dish, the heap of rice, in snowy perfection is the pride of the good housewife.

The reward of labour is, perhaps, in this case not merely æsthetic. Every step in the primitive domestic process of preparation of rice for food renders easy the detection and removal of abnormal or discoloured grains, and reduces proportionately the chance of consumption of disease-producing elements. The probability of any grain recognisably bad entering into the food is reduced to a minimum by the various pickings over and washings.

Were beri-beri due to any coarse *parasite*, resembling, for instance, *ergot* or smut, it might be expected that natives whose rice as grown was infested should, nevertheless, be preserved from its operation in food through their careful selection of the grain before cooking it.

But this is not all. If the disease were due to any form of *saprophyte* not originally found, or only sparingly found, in fresh padi, but perhaps secondarily attacking decorticated stored rice, then the custom of preparing at one time only so much rice as is required for daily use would also be salutary and protective, saving its consumers from the effect of poisons the result of saprophytic growth.

The use of such *fresh* rice, as it will for convenience be referred to here, is in the Malay Peninsula confined almost entirely to the Malays, and, it will be seen later, has an important bearing on the distribution of beri-beri among them.

In Burmah, padi is husked or shelled by being passed between a pair of small millstones or rollers of hard wood, grooved on the

working face, and set at such a distance apart as serves to split off the husk by friction without breaking the grain. The removal of the pericarp is then effected by pounding with wooden pestles in mortars of either wood or stone.

In China, in Cochin-China, in Siam, in Burmah, in Borneo, in the Dutch Indies (Java, Sumatra, the Celebes, etc.), in Madagascar, wherever the peasantry live upon grain grown by themselves, similar methods of preparing fresh rice are in vogue.

II. *Uncured Rice, Stale White Rice.*—The bulk of the world's supply of rice, grown in the great valleys of the Irawadi and the Menam, is husked in mills on a large scale by methods which imitate, or rather are an extension of, the manual process, but yet differ from it in essential and perhaps all-important particulars. The crops gathered by natives, for the most part by hand, collected at innumerable points over vast areas of country, finally arrive, by many channels of jungle-path and road, of canal and river, at the centres, generally ports, at which the mills are situated.

At the mills the grain is thoroughly dried, sifted to remove all foreign bodies, husked, hulled, or shelled, winnowed and screened, 'scoured,' 'polished,' and rewinnowed, 'ended,' and 'sized.'

Husking by machinery involves the removal of husk and true pericarp both together, of the episperm with the latter, and often much, or even all, of the gluten layer of the seeds also.

The grain is delivered on to 'shelling-stones' of about 6 feet in diameter, set the full length of a grain apart, and revolving about 120 times a minute. By these it is hulled.

The completely stripped grain, further screened and winnowed, and consisting of nothing more than the endosperm of the ovary with its gluten layer, is next 'scoured' by a process of what is called 'barley-milling.' The scouring machine consists of one or more stones—generally of sharp-grained sandstone—set on a spindle, revolving inside a fixed casing at a speed of from 120 to 250 revolutions per minute. The interval between casing and stone may be 12 to 16 millimetres ($\frac{1}{2}$ to 1 inch).

The casing is of fine wire-netting or gauze, supported on metal or wood. Through this machine the decorticated grains are passed several times in order to remove the small particles of dust adhering to them, and to *smooth away irregularities of surface*. The dust is driven out through the fine wire screen. *The whole of the gluten layer is removed in this process.*

A further course of friction between a fixed wire screen, of conical shape, and a cone covered with long-wooled sheepskin, revolving 200 times a minute, inside it, 'polishes' the grains. The removal of their points is termed 'ending.' They are last 'sized' through screens.

The processes thus employed effectually attain the end aimed at. The clean white rice delivered is smooth, rounded, uniform in aspect and in size in different samples. But this commercial success is achieved at the cost of hygienic disaster. Directed by nothing more intelligent than a desire to appeal to the eye of the customer, the commercial treatment of rice results in products the nutritive value of which is in exactly inverse proportion to their market price. For the more the grains are submitted to fancy processes of scouring and polishing, the more complete is the removal of all the gluten layer, which most requires preservation; and the less thoroughly the grains are decorticated the greater is the chance of their retaining this, their most valuable constituent.

And, finally (although this must be stated less as a fact than as a speculation), whatever infectible products (spores) there may be among any of the grains, are in the multiplication of milling processes and the contact of grains with each other more thoroughly rubbed in to each individual grain and distributed among all.

Rice prepared by such dry-milling processes is known under various market designations, but all the samples, agreeing in general characteristics, may be included under one term. Because they differ from the sort next to be described, mainly in the special feature that the grain from which they are prepared has never been submitted to heating in water, whereas the next sort is, I shall in the sequel refer to all rices prepared wholesale in mills by an exclusively dry process as 'uncured' rice.

The grain from which uncured rice is made is dried, and kept dry as far as possible, before it is consigned by the local husbandmen to the mill agents. But during its transit, heaped in bulk, whether loose or in bags, down river to the mills, it has chances of exposure to moisture and fermentation, which are probably only too often incurred, and which must be prejudicial to its soundness.

No minute inspection is made—dealing with such thousands upon thousands of tons of rice as are annually passed through the mills, it is not possible that any could be made—of the separate grains, or even of small parcels of the grain. There is every

probability, therefore, that diseased samples, if such occur, pass into the masses of rice sold commercially, wholly undetected.

Moreover, the possibility which there is, even in the carefully-conducted separation of his rice from the raw grain by the Malay, that poisonous matters derived from hull and pericarp (if present on them when raw) should contaminate the separated seed, is, in the commercial modes of preparation, much increased. Not only are the outer envelopes minutely ground up and intimately mixed with the rice by heavy attrition, but there is no washing of the product immediately after separation of the seeds from the envelopes, as when the householder prepares his daily store. As a fact, the rice sold commercially as white rice, whether coming from Siam, French Indo-China, or Burmah, invariably shows fine, almost impalpable, powder clinging to and spread among the grains.

Upon every showing, therefore, if there be any poisonous agent which occurs on or in raw rice as harvested, that agent must be more plentiful in the commercial dry-milled grain than in fresh rice, or in rice prepared by another method to be described, in regard to both of which the processes employed must, to some extent, serve to eliminate it.

Deprived of their envelopes (and especially the surface layer of the endosperm), their natural protection, the stripped and bare and bruised rice-seeds rapidly decay. Although, when carefully stored as *padi*—being, above all, kept dry—the seeds of this, like other cereals, will preserve their vitality for an indefinite time—certainly many years—the process of hulling, probably as the direct result of the violence inflicted, rather than the exposure to atmosphere, entails immediate destruction of the germinating power of the seed. Decorticated rice is, in fact, devitalized—dead—rice. The grains in this state represent nothing more than so much organic matter, friable and hygroscopic, an excellent medium for saprophytic growth.

III. *Cured Rice*.—In India, though not in all parts of it, a different method of making rice from the *padi* is pursued.

There, following immemorial custom, taught by a wisdom based, perhaps—who knows?—upon a certain, if empirical, knowledge of the cause and prevention of the very disease now under discussion, the native first soaks, then *boils*, and next dries the raw grain before husking.

‘In Bengal,’ Major Buchanan says,¹ ‘the *padi* (*dhan*) is well

¹ In a letter to the writer.

soaked in water for two or three days before binding, and then spread out on the ground to dry, before it is sent to the *denkhi* or mill for husking. Now, this boiling in big iron pots would possibly kill any fungus growth if the rice were diseased, *à la* pellagra. . . .¹

The following is an official account by Major Buchanan of the 'method of making rice from padi' in the Bengal gaols:¹

'The padi is first soaked in water for about twelve hours (one night). The soaked padi is then put in earthen or iron vessels, and about half the quantity of water added (rice 3 parts, water 1 part), and heated on a slow fire till the grains are burst.

'The water is then strained off, and the boiled padi is spread out in the sun to dry. It is spread out on clean ground or on platforms, and constantly turned over by the hands or feet. The most important part of the process is the drying. When properly dried the rice should easily separate from the husks by rubbing it on the ground with the bare heel or on the palm of the hand.

'The dried rice is then taken to the *denkhi* (mill), and winnowed to remove the husk.'

This is the detail followed in Government gaols. The native, as observed above, generally soaks the grain in cold water for several days before boiling. The removal of the husk only, leaves the dense, dark-red, closely-adherent pericarp still attached to the grain. If this is desired to be removed, as it usually is, for rice used by the well-to-do, it may be done in the usual manner by pounding by hand in the wooden mortar, or by milling.

The result of the maceration—the 'curing'—having been, as explained above, to cause the aleurone layer formerly cleaving to the woody pericarp now to adhere more closely to the endosperm, pounding and lightly milling boiled grain results in the removal from it only of the pericarp, but not of the aleurone (gluten) layer.² Consequently, this sort of rice possesses a far higher nitrogenous value than any sample prepared and thoroughly scoured by dry-milling processes. It has the food value of 'red rice,' without the disadvantages attaching to the presence of the somewhat bitter and undigestible woody parts retained in that sort of rice, and, according to the analysis given by Vorderman, cured rice would therefore be as a food better than some samples of wheat.

¹ Communicated to the Government of the Straits Settlements by the Government of India. Corresp. Sec. to Government of India. No. 337, 1/11/02.

² Professor Marshall Ward who with great kindness examined a sample of ordinary cured Indian rice for me, stated that 'in all the grains of decorticated rice examined the aleurone layer is left behind on the grain.'

Another result of the retention of the aleurone layer must be that the endosperm is to some extent protected by it against decay. But, as I have seen samples of cured rice very sour (owing to their never having been properly dried), I am not inclined to attach much importance to the protection which this thin stratum can afford.

The process of 'curing' has a yet more important result than either of these.

It is hardly necessary to labour the point how effectually, as Dr. Buchanan suggests, such treatment of the grain must dispose either of preformed poisons, soluble or otherwise, attached to husk and pericarp, or of ferments or organisms which would thus be made first to germinate, and then, in that vulnerable stage, be destroyed by heating. The process is, roughly, and under open-air conditions, in fact, such as is daily resorted to in every laboratory to sterilize, or render free of noxious germs, various materials which may contain them.

Prepared by such a method, it is obvious, rice derived from however contaminated a raw grain originally would initially be incapable of conveying much, if any, poison to its consumers. Whether and to what extent it might become changed or fermented subsequently would depend upon the manner in which it was stored and the fresh germs to which it should be exposed.

It would seem that the Indian method of treating the grain may result also in partially 'malting' it. The soaked and boiled grains are certainly changed in aspect and physical constitution, appearing yellower, tougher, and more dense. They require longer to cook, but appear to be more rapidly digested.

Rice of this sort is also prepared wholesale in mills.

I do not know whether all the rice of this class prepared in and exported from Bengal is native-grown, but in the Straits Settlements much of the rice sold as having been so prepared is locally produced Kedah- or Malacca-grown. Raw padi is also imported from other places to be treated locally in the same way.

Indians (Tamils) term rice made from boiled grain, whatever its source, *pulunga arisi* (boiled rice). In this work all rices of this kind will be referred to as 'cured' rice.

Recapitulation.—To recapitulate, the distinctions in the qualities and potentialities for becoming toxic of the various sorts of rice in consumption, classed according to the modes in which they are dressed, are as follows :

I. RED RICE.—Retaining all or most of the pericarp. In such grain the whole of the gluten is also preserved. It is thus far more nutritious than white rice; any preformed poison it may contain is *relatively* less in quantity, *ceteris paribus*, than in other rices; and the surface being unbroken, it is better protected from invasion by saprophytes—decomposition—or by any specific growth; last, it is generally fresh.

Red rice is but little used—indeed, seldom met with—in the Federated Malay States, in China, or in India, or in Ceylon. It is extensively used, however, in Java, and has there been made the subject of special investigation by Vorderman, with results which will be detailed later.

II. CLEANED OR WHITE RICE.—According as more or less of the pericarp is removed and the rice is made more nearly clean and white, proportionately the amount of gluten-layer removed is increased, the proteid value is diminished, the quantity of preformed poisons (if any present) in the seed becomes relatively larger, and the liability to saprophytic decay or invasion by specific ferments or growths is rendered greater.

Cleaned white rice appears under three aspects:

1. FRESH RICE, *usually locally grown*, is

(a) *Fresh*.—Used as newly prepared from the padi, it presents the character of the seed unaltered.

(b) *Selected*.—Washed, and picked over carefully, it is freed of all adventitious bodies and obviously diseased grains.

(c) *Unscoured*.—Retaining much of the gluten layer, therefore of good food value.

2. UNCURED RICE, *usually imported*, is

(a) *Stale*.—Exposed for periods more or less prolonged after cleaning, the seed, under the influence of air and moisture, is altered in character.

(b) *Unselected and unwashed*.

(c) *Scoured*.—The gluten layer almost or wholly lost, it is of lower nutritive value.

(d) As a result of the preceding conditions, it is more liable to be invaded by saprophytes (producing common decay) or any special ferments or organisms which, occurring in the husk or in only single or scattered grains before milling, are distributed freely over all of them in that process.

3. CURED RICE, *made from local grain, or imported*, is

(a) Either fresh-cleaned, or stale—commercially always stale.

(b) *Sterilized* by boiling while in the husk, therefore *protected*

from the possible spread of any specific organism originally present on or in any of the seeds, which might render it poisonous.

(c) *Unscoured*, and retaining the whole of the aleurone layer, it is highest in food value.

It is upon one or another of the three principal sorts of white rice which have been described—the *fresh*, the *uncured*, and the *cured*—that natives all over the East, and certainly in British Malaya, subsist. Upon which of them is eaten, it will be seen, depends the liability of the consumer to beri-beri.

Proof of the Connection between Beri-beri and Uncured Rice.—

It may seem to some that no observations to determine the causal connection of a given agent with a particular disease can be satisfactory, based upon facts not the result of predetermined conditions, of directly controlled experiment. But not all experiments are uncontrolled, the conditions of which are not laid down directly by the scientist; not all observations are worthless which are not conducted in the laboratory. Great experiments are daily in operation naturally, from which it requires but care to draw valid conclusions.

Suppose it were possible, in a country in which beri-beri was known to be endemic, to introduce a large number of individuals all susceptible to, but all previously free from, the disease, or from contact with cases of it, and to domicile them in the country each for two years or more.

Suppose, next, that the experiment were tried of restricting one section or class of the immigrants to the use only of the one, the cured, and the rest—it may be a larger section—to the use only of the uncured sort of rice.

Suppose, again, that to avoid such fallacies as might attach to results obtained in restricted or special localities, the observations could be carried out over a really large area—say 10,000 square miles; that, for similar reasons, the numbers dealt with should be also large—say 50,000 persons or more in either section.

Let all these individuals be scattered, as far as possible, uniformly, or at least without selection of locality, throughout the area of observation; let no discrimination be made in their occupation, and let, in fact, all the circumstances of their environment be left as free from control, and as nearly equal, therefore, as possible.

Let, finally, the observation be continued over such a period of time (say twenty years) as shall completely obviate all sup-

posed seasonal or even multiannual and economic factors of variation.

Would not an experiment made under such conditions be for the purpose as suitable as—it would certainly be more extensive than—any which could be devised in a laboratory?

Suppose that, as the result, it were found that among the persons fed upon uncured rice, beri-beri extensively, always, and everywhere prevailed: but that the people eating only cured rice remained always and everywhere absolutely exempt. Would not such an observation be regarded as proof sufficient of the theory that the disease depended on the one sort of rice?

If, finally, the converse of the experiment, so far as the feeding of the two classes put under observation was concerned, could also be carried out, and if, as the result in this case, it were noted that the people, untouched on the cured, became equally affected with others when fed upon the uncured grain, would not the proposition be regarded as completely proved?

Now, precisely the conditions here supposed have actually been realized. An experiment on the gigantic scale contemplated has been made—is, indeed, still going on—the result of which establishes exactly the conclusion indicated.

There may now be detailed, seriatim, the actual data upon which this conclusion has been based.

Evidence from the Chinese and Tamil Immigrants in the Straits Settlements and Neighbouring Native States.—1. The *field of experiment* has been the colony of the Straits Settlements, with the neighbouring native States, on the western side of the Malay Peninsula. The latter have been under British protection during the last thirty years, and have been opened up and developed, especially during the last two decades. The extent of this field is over 13,000 square miles. In its physical geography the area comprises features of every degree of diversity. In climate its different portions show but little variation. The rainfall averages 100 inches. The mean temperature is about 80° F.

2. The *period of time* dealt with will be limited (as far as the citation of statistics is concerned) to the decade 1891 to 1901, during which the country and its inhabitants have almost everywhere been under medical supervision.

3. Throughout the whole area *beri-beri* has been and is endemic. Figures already cited (p. 2) show the distribution of the disease from 1891 to 1901.

4. The *rice*, forming the staple food of the immigrants, has been of the two sorts distinguished, from the mode of their preparation, as 'cured' and 'uncured' respectively.

In the native States of the mainland and in Province Wellesley the padi grown by the Malays suffices for their own consumption,¹ but elsewhere and for the rest of the population the whole of the rice consumed is imported.

The chief sources of supply of uncured rice are Siam, French Indo-China, and Burmah. Locally, these supplies are recognised as *bras* (rice) 'Rangun,' or *bras* 'Siam.' The former is usually slightly the cheaper, but I cannot find that those accustomed to either one of these two varieties object to the other upon occasion, and all the uncured varieties may be treated as one sort, and will be referred to together.

The Indian or cured rice was until recent years always imported from India, chiefly from Calcutta, and was known as *bras* 'Bengal.' Alterations in freight and exchange made the price of this rice so dear, however, that endeavours to meet the demand for it were made by the establishment of local mills and factories, in which grain grown locally could be treated after the Indian manner. The rice so prepared locally is known as 'Singapore'- or 'Penang-boiled,' and being much cheaper, has almost entirely displaced Bengal rice in the dietary of those formerly using only the latter.

It is upon one or the other of the two sorts of rice which have been described that the whole of the immigrant population of the beri-beric area now under examination has, for the last twenty years, been fed.

5. Into the beri-beric area there have been introduced over 150,000 immigrants, all susceptible to, but previously entirely free from, beri-beri. The immigrants are chiefly Indians and Chinese.

There is a vast yearly traffic of persons to and fro between the British Malayan areas and other countries, especially China and India. Both Chinese and Indian (Tamil) coolies are imported under contract, or come independently, in large numbers to seek work and fortune in mining or agricultural enterprises. The number of those returning annually is also large. Both because of this emigration, and because the death-rate everywhere in Malaya, among both these races, greatly exceeds the

¹ In Province Wellesley and certain parts of Perak and Malacca sufficient is grown for export as padi.

birth-rate, their numbers have to be constantly recruited by the importation of fresh adults.

The Chinese immigrants are, through one cause of selection or another, almost invariably healthy young males under twenty-five. The Tamils recruited to work upon the estates, and at other works, are examined especially with a view to their physical fitness, and although strong labourers are not always secured, the actually sick are always excluded.

Account is kept by Government officials of the numbers of immigrants and emigrants, from which the facts mentioned here are taken.

The Chinese contribute the largest proportion of each class absolutely and relatively of those who settle permanently in the country. Every year some 100,000 of this race arrive, and about three-fifths of that number return to China. The Census Report of the Federated Malay States for 1901 states (pp. 81, 82) that between 1891 and 1901 there entered into the two States Perak and Selangor 887,900 Chinese, and emigrated again 652,652.

Immigrants from India, chiefly Tamils from the South, numbered, from 1880 to 1890, 159,313; emigrants, 103,413. For the decade which we are considering the immigrants were, 215,020, the emigrants returning 142,093.

During this period, while the total population, including all races, has increased from 545,510 to 691,088 individuals, the increase among the Chinese has been 133,156, that among Tamils 21,729. (In these returns *male adults only* are referred to.)

These numbers afford an amount of material upon which to base statistics as to the relative incidence of disease to which exception cannot be taken, at any rate upon the ground of insufficiency.

As regards the susceptibility of the two classes of immigrants, Chinese and Tamil, to the influence of beri-beri, and of their previous entire freedom from it, an observation already made may be recalled. The first experience which hundreds of thousands of coolies, coming from all parts of China, have of the disease is gained after their arrival here. There is no colloquial specific name for the disorder, I believe, in their language. Their susceptibility to it is a matter unfortunately too notorious.

As regards the other class of immigrants—the Tamils—almost the same observations apply to them. 'Beri-beri is,' Dr. Buchanan,¹ for instance, says, 'unknown in Bengal.' It is a rarity

¹ *Lancet*, August 27, 1898, vol. ii., p. 577.

in the Deccan, whence the Tamils who come to this country are recruited. It was among Indians, however, that Malcolmsen,¹ one of its earliest describers, first observed beri-beri. Outbreaks are of frequent occurrence among lascars in the Indian marine, and have been reported and discussed by Anderson.² In Rangoon it was noted by Barry³ that the class affected were almost exclusively Hindus, the majority of whom were Tamils, immigrant from Southern India, precisely the people who, we shall presently see, escape it in Malaya. There need be no doubt, therefore, it is worth while insisting, as to the susceptibility of the Tamil.

6. *One class of these immigrants has been fed exclusively upon one—the uncured—sort of rice; the other class has fed as exclusively upon the other—the cured—rice. The Tamils use the cured, the Chinese the uncured grain.*

It may seem to the reader that to affirm an exclusive addiction on the part of these two classes to the use of the two sorts of rice described respectively is too strong a claim to make; but to those acquainted with the circumstances, those who know, among other things, the inveteracy with which Eastern races cling to established habits, there will be nothing astonishing in the statement. The Chinese in Malaya eat only uncured rice, because it nearly resembles in flavour and in digestibility that to which they have always been accustomed. It is everywhere easily obtainable, and the varieties of it sold are, moreover, the cheapest of the rices in the market. The cured rice, when they are obliged to take it, as when I have given it to patients experimentally, seems to them of poor flavour, and is, they declare, unsatisfying; it produces dyspepsia, and nearly always, for the first few days of its use, diarrhoea. There is no reason, therefore, why they should ever use the cured rice, and, in my experience, they never do.

The same statements hold good, but apply with even increased force to the Tamils. The supply of this class of labour, in demand for agricultural enterprises and undertakings for which Chinese labour is deemed less suitable, or found too dear, has always been short of what is required. In consequence, and because also the Indian Government insisted upon attention being paid to the customary needs of coolies recruited from their territory, employers of Tamil labourers everywhere, throughout the period

¹ 'A Practical Essay,' etc., 1835.

² *Ind. Med. Gaz.*, September, 1901.

³ *Ibid.*, September, 1899, and May, 1901.

and area now under consideration, have made a point of studying their needs, and of securing for them rice of the kind, and as far as might be of the quality, to which they had always been accustomed. When Bengal rice became too dear for importation, as has been seen, a local rice, prepared in a similar manner, was put upon the market, which meets, so far as I can ascertain, all the demands made upon the other. If not absolutely every Tamil in the country uses this—the cured—rice, yet certainly all ‘Statute Immigrants’ and estate coolies do so, and among ‘free coolies’ it can confidently be affirmed, those who do not are so few that their numbers will not affect any result or any conclusion to be drawn from the habit of the majority. Indeed, the rare exceptions which are to be noted afford, as will be seen, proofs in regard to the theory under discussion of exceptional value.

The two classes of immigrants under consideration, it is to be remarked, offer no particular differences in regard either to the localities in which they are employed or the labour upon which they are engaged. Although they seldom inhabit actually the same buildings, they live in close contiguity. In trade they mingle freely together, and at labour they are employed upon the same tasks, as estate work, road and railway making, and (latterly) even mining, a pursuit formerly confined entirely to the Chinese, and believed by some to have an etiological bearing on beri-beri.

But from one cause and another it does happen that the Chinese as a class are rarely underfed or destitute. In spite of their addiction to the opium habit, moreover, which itself aggravates, and often indirectly entails, much of the sickness to which they succumb, they are far less affected by diseases generally than are the Tamils.

In Negri Sembilan, for instance, in 1901 the Chinese, forming 351 per 1,000 of the whole population, contributed 338 per 1,000 of all deaths registered, and 520 per 1,000 of all the admissions to hospital. Yet the admission-rate per 1,000 Chinese living was for that year only 89.

But in the same year the Tamils, forming only 56 per 1,000 of the population, contributed 84 out of every 1,000 deaths registered, and 454 out of every 1,000 admissions to hospital. Their admission-rate per 1,000 living was 395.

The death-rate annually per 1,000 living was 36 for Chinese, 116 for Tamils.

In Perak, in 1899, the Tamils, estimated to form 56 per 1,000 of the population, contributed 98 per 1,000 of all the deaths

registered, and 254 per 1,000 of all admissions to hospital. The number of these admissions per 1,000 living annually was for Tamils 163, while for Chinese it was only 110.

In Selangor, the same year, the proportion of Tamils in the population being 83, their contribution to all deaths registered was 89, and to hospital admissions 204 per 1,000. Their admission-rate to hospital per 1,000 living annually was 302, that for Chinese being 86.

The death-rate among Tamils in Selangor in 1900 was estimated by the State surgeon as being 136 per 1,000 annually.

It is clear from what has been said that the Tamil, in this country at least, is far sicker than the Chinese. He has four times as much sickness, and five times as great a mortality. This fact will heighten the significance of the statement which follows, that—

7. *Among the Chinese—the users of uncured rice—beri-beri is everywhere prevalent, whereas the Tamils—the users of cured rice—are everywhere and always completely exempt.*

The former part of this statement, unfortunately, needs no more proof; it has already been shown how large a proportion of Chinese succumb to the disease every year.

The extraordinary freedom of the Tamils from beri-beri has only lately been made conspicuous.

In the earlier days most of the Tamil coolies imported to labour in this region were, and even now many are, engaged under the terms of a special ordinance, the stipulations of which, made at the instance of the Government of India, prescribe careful attention to their sanitary and other wants. Medical supervision is everywhere provided for these, as they are termed, 'Statute Immigrants,' and record is kept by agents appointed by Government of the numbers engaged, of the births, deaths, desertions, sickness-rates, and other facts relating to them, which is published annually. No other class of the population has so much attention paid to it, and of none are the ailments and maladies so carefully watched and so scrupulously, if not always accurately, diagnosed and reported. It is impossible, therefore, were a disease as easily recognisable as beri-beri to occur among them anywhere, that it should escape notice.

Rather is it probable that other affections should sometimes be mistaken for a disease so common.

Analysis of the reports of the Indian Immigration Agent to Government for the years 1891-1901 shows that (see tables, pp. 158, 159).

During the ten years the number of individual coolies imported as statute immigrants to work on estates in the Straits Settlements and Federated Malay States was 26,952.

The average annual Statute Immigrant population of the estates was 6,753.

The total admissions to hospital were 79,871.

The average annual admissions to Estate hospitals for all causes were 7,987.

The actual number of deaths recorded was 2,423.

The *sickness-rate* was thus 118 per cent., and the *mortality* from all causes among the whole population 35 per 1,000.¹ The *case-mortality* for all diseases together in the hospitals was 68.4 per 1,000, showing that the conditions for which admission was sought were grave. Among the causes assigned for the 2,423 deaths recorded, I find no less than fifty-six different morbid conditions named—proof enough, were any needed, that the diseases were carefully diagnosed.

Here, then, was a population of more than 26,000 individuals, of all ages and castes and both sexes, scattered through a region in which beri-beri was endemic, employed at every kind of labour, and exposed to every climatic condition to which others—Chinese—were exposed, brought under observation for ten years. Although they were less sickly, showed lower mortality as a body than free coolies of their race, their sickness-rate (118 per cent.) and death-rate (35.6 per mille) were yet high.

The diseases to which they did succumb were numerous, and had they been Chinese, it is certain that beri-beri must have figured largely in the list.

But the fact, sufficiently astonishing, is that, so far from being an important or even a common cause of sickness, beri-beri, if it ever occurred among these coolies, did so with the extremest rarity, and it may be said to have been non-existent.

In no one of the ten annual reports² dealing specially with them is it ever mentioned anywhere as a cause of sickness. In the records of two separate years only, out of the whole ten, is the death of any coolie even *ascribed to* beri-beri: once, of a single coolie, in 1892, in Selangor; again once, also of a single coolie, in 1900, in province Wellesley.

¹ The mortality, high as it was, among these statute Indian immigrants was yet far lower than among free coolies of the same nationality at the same time and in the same districts.

² Of the Indian Immigrant Agent to the Government of the Straits Settlements.

**Analysis of all Causes of Death amongst Statute Indian Immigrant Coolies in the Straits Settlements
and Native States during Ten Years.**

Disease.	1891.	1892.	1893.	1894.	1895.	1896.	1897.	1898.	1899.	1900.
Abortion, child-birth, and puer- peral complaints	12	2	4	...	4	...	1	5
Accidents, surgical operations, and injuries	9	2	...	6	6	2	2	2	1	2
Anæmia and debility	76	141	1	68	68	29	63	34	49	87
Apoplexy (cerebral hæmorrhage) and tumour in brain...	2	...	2	4	...	3	1
Albuminuria	1	1	...	3
Angina pectoris	1
Ascites and cirrhosis of liver	...	1	...	3	1	1	...	3	1	...
Asthma	1	...	1
Ankylostomiasis	2	1
Beri-beri	1	1
Bronchitis	3	1
Cholera and choleraic diarrhoea...	12	1	...	2	3	3	9	1	2	1
Colic	2
Convulsions	2	1	...	1	...
Cancer
Cellulitis	1	1
Cystitis
Diarrhoea	115	152	46	69	83	57	42	90
Dropsy, 'Endemic'	7	11	3	77	3	2	...	1	1	...
Dysentery	86	57	30	33	11	42	51	43	34	58
Dyspepsia	1	1	1	1
Epilepsy	1	1	...	1
Enteric fever	3	...	4

DISEASE VARIES WITH SORT OF RICE

159

	14	8	...	9	2	4	8	4	172	158	276
Fevers ...	14	8	...	9	2	4	8	4	172	158	276
Gangrene ...	1
Heart disease ...	2	2	...	5	2	3	1	3	4
Hydrophobia	1
Intussusception of bowel	1	1	1	2	1
Melena	1	...
Measles	1	1	1	...
Old age	5	4	2	1	...	5	3	7
Phthisis ...	4	2	...	6	3	18	4	...	4	2	5
Pneumonia ...	14	13	...	1	1	...	1
Pleurisy
Paralysis ...	1	1	2
Peritonitis ...	2	1	1	2	2	...
Pyæmia	1	...	1
Pelvic cellulitis	1
Phagedæna	4
Poisons ...	1	1	1
Rheumatism	1
Small-pox ...	1	1	1	...	1	1	...
Septicæmia	2	...	2	1	...	2	4	...
Scrofula	1	...	1	5	2	1	...	2	...	2
Splenitis	1
Sunstroke	2	1	...
Tetanus ...	1	1	2	...	1	1	1
Undiagnosed and unspecified ¹	159 ¹
Ulcers ...	4	7	1	1
Venereal ...	4	4	1	...	1	2	...
Total ...	373	410	193	234	170	195	242	172	158	276	2,423

¹ In the year 1893 the usual form of returns was for some reason not completed.

160 THE CAUSE AND PREVENTION OF BERI-BERI

Yet in the same area in which these coolies were placed, and during the same period, one-fifth to one-third of all the deaths of Chinese in hospitals were caused by beri-beri, and probably always two-fifths of their gross mortality was due to it.

At Caledonia, an extensive sugar estate in Province Wellesley, almost all the coolies employed are Tamils on agreement. The rice given them is exclusively cured rice. At the estate hospital during the last eleven years 21,487 of these Tamils were treated as in-patients, of whom a single one only (in the year 1900) was stated to have had beri-beri!

In the province in which the estate is situated beri-beri, if less prevalent than elsewhere, is still a conspicuous disease among the Chinese, most of whom are employed upon sugar estates, or as padi planters.¹ Thus at three Government district hospitals the proportions of them admitted with beri-beri² were 37, 84, and 178 per 1,000 respectively. At Bukit Mertajam 140 cases were treated among 3,702 patients; at Butterworth, 187 cases among 2,197 patients; and at Sungei Bakap 650 cases among 3,634 patients during the last two decades.

Another instance not less significant is that of the railway base hospital, at Kajang in Selangor, during 1900-1902, at which Tamil coolies imported to work on the railway were treated. Sickness was very prevalent among them, but of the 5,576 cases treated in hospital none had beri-beri.

Yet at the district hospital serving the same area in which these coolies worked two-fifths of the Chinese admitted during the same years had beri-beri (737 cases in 1887 admissions for all causes).

The Tamil railway coolies were also supplied exclusively with cured rice.

The last two instances given are the more noteworthy in that there is no likelihood of there being any inaccuracy in the results presented. At both the Caledonia estate and the Kajang railway works all the coolies were under the direct care of experienced European surgeons.

¹ There is no protection derived from working in padi or sugar. In 1881, and again in 1885, severe epidemics were reported of beri-beri among Chinese coolies. That in 1885 was stated to have been 'entirely confined to the Chinese Sinkhehs working on different estates in the province' (Straits Settlements Blue-Book, 1885).

² I.e., the proportion of Chinese admitted for beri-beri to all Chinese admitted to hospital.

Too much stress can hardly be laid upon the significance of these records. That this population of sickly Tamils, who have no natural immunity from beri-beri, should have remained entirely unaffected by it for a space of two decades, in a region in which it was everywhere a constant cause of sickness and mortality among their neighbours the Chinese, is extraordinary, and could not be credited as possible were the cause of the malady anything of the nature of a miasm or an infection.

But the facts are beyond dispute, and justify the statement that the Statute Immigrant Tamil coolie population is, at least in Malaya, exempt from beri-beri. *They are the people who eat exclusively cured rice.*

In dealing next with *free* Tamil immigrants, it is first to be observed that there are factors, some of which will tend to make the actual, and others the reported, occurrence of beri-beri among them more frequent than among Statute Immigrants.

1. In the first place, there are two sections of Tamils in whom that conservatism in regard to the sort of rice eaten, so rigorously preserved among the statute coolies, both through their native habit and Government insistence, tends to break down. They are the extremes of the Tamil social scale. A numerous section is composed of the traders, clerks, and other persons above the labouring class, who are specially inclined, with increasing 'education' and advancement in circumstances, to throw off habits essentially of their caste and race, and many of these individuals eat any of the varieties of rice accessible, although upon inquiry they usually admit a preference for the cured sort.

The others are the indigent—beggars, whom their circumstances compel to eat any rejected scraps of food, others whom poverty obliges or thrift induces to buy the cheapest, the uncured sorts. There is now quite a large and settled population of Tamils in the country, an increasing proportion among whom are adopting wholly the customary rice of the country for their food.

It must follow from these facts that the proportion of Tamils suffering from beri-beri among the free will be greater than that among the Statute Immigrant coolie population. The latter may be regarded as wholly protected against the disease. But it is to be expected that a few cases of it should occur here and there among the former.

2. In relation to free, equally with the Statute Immigrant coolies also, certain sources of error in the ascription of maladies

162 THE CAUSE AND PREVENTION OF BERI-BERI

incurred to their true cause will exaggerate the apparent incidence of beri-beri.

There is a disease very common among Tamils everywhere in the native States, the principal features of which are great anæmia, accompanied by weakness and its other natural sequences of symptoms, and almost invariably extensive general anasarca. Anæsthesia is not found in them; there are no paræsthesiæ or allochiria, no tenderness or subsequent atrophy of muscles, no true paresis. Organic cardiac, renal, and hepatic lesions are usually absent; so also are ankylostomata.¹

Their condition does not appear to depend on antecedent malaria,² and seems to me to constitute a specific syndrome of symptoms possibly related to the 'epidemic œdema' described elsewhere. The disease is certainly not beri-beri, but—this is the point I wish to insist upon here—it may easily be mistaken by the inexpert, or when careful examination of the nervous system is not made, for beri-beri. To such an error in diagnosis it is likely is due the inclusion in the returns of some cases as beri-beri which really were not so.

3. Lastly, the statement made, that *so long as they keep to the habits of their class in regard to rice*, the Tamils in Malaya do not get beri-beri, is not invalidated by the inclusion in returns of cases of beri-beri occurring among Tamils *by whom this habit was not adhered to*, or in regard to whom this point was not decided. It happens to be a fact that in all of the cases—some eight or ten—of the disease in this race, which have come under my own observation, the habit of eating uncured instead of cured grain, and that almost always for several months prior to the onset of the complaint, has been clearly established.

Yet, in spite of all the influences detailed, which tend to exaggerate the apparent frequency of beri-beri among Tamils, as a matter of fact the free Tamil has a freedom from beri-beri, compared with the other races among which he lives, nearly as complete, according to records, as is that of the Statute Immi-

¹ I say this with all due deference to an opinion on these cases expressed to me by Dr. Daniels (who saw some of them in my wards) to the effect that they were ankylostomiasis. To avoid possible misconception, I should perhaps qualify this statement by saying that ankylostomata are rarely in these cases present in such numbers as to justify the belief that the symptoms noted were due to them. On the contrary, few such cases show many ova or persistently show ova in the stools. In the majority very few; in many cases none at all are to be found.

² Or dysentery.

grant coolie; and when allowance is made for the sources of error mentioned above, the statement that *so long as he eats only cured rice* he is entirely free from beri-beri may be accepted as true for him also.

At the Seremban (general and pauper) Hospital, in the ten years 1891-1900, there were admitted 7,884 Chinese, of whom 3,429, or 434 per 1,000, had beri-beri; and 3,368 Tamils, of whom 16 only had beri-beri, a proportion of 4 per 1,000. Among the 16 cases inquiry as to the kind of rice eaten was made only of the last 6. All these, it proved, had been addicted for considerable periods prior to their illness to eating uncured rice, as it may fairly be surmised had been the other ten also.

Excluding the 6 *certain* uncured rice-eaters, we have a proportion of 3 beri-berics only among every 1,000 Tamil coolies treated at this hospital during ten years. Such a proportion, compared with the relatively enormous incidence of the disease upon the Chinese—434 per 1,000—alone is a demonstration of the practically complete exemption of the free Tamil coolie from beri-beri; and the positive evidence as to the presence of the habit of eating uncured rice in the cases—one-third of all those recorded—in which this point was ascertained leaves little room for doubt that *among the independent coolies eating only cured rice in Negri Sembilan*, as among statute immigrants generally, *the exemption from beri-beri is absolute*.

In regard to the other States of the Federation, I have been unable to collect statistics as complete, but those of the few years for which I have been able to obtain them are no less convincing.

In Perak in 1899—Hospital Admissions.—*Perak* is the largest and most numerously populated of the States composing the Malay Federation on the west side of the Peninsula. Returns of all the (eleven) large hospitals in which pauper and other patients are received, with which I have been furnished by the kindness of my colleague, Dr. Wright, the State surgeon, show that there were treated during 1899 for all causes 19,693 patients, almost all of these being coolies. Of these, 2,817 were admitted for beri-beri. Upon a basis of numbers so large as this, it must be conceded, a really reliable estimate of the relative incidence of the disease upon different classes can be made.

Analysis of the figures given shows that of all the admissions Tamils formed 5,019, or 254 per 1,000, and of admissions exclusive of beri-beri 297 per mille; but of admissions for beri-beri they

formed only 2 per 1,000. Actually, of the 2,817 cases of beri-beri admitted in that year, 6 only were Tamils.

In these hospitals not a single Tamil died from beri-beri.

Thus, at a place and time when the admissions for beri-beri among the Chinese amounted annually to 172 per 10,000 of the adult male population, and the death-rate from it to one-third of that amount, its incidence upon Tamils was but 4 per 10,000 living, and the mortality from it absolutely nil.

'Figures,' it is a saw, 'may be made to prove anything.' But it must be conceded that little making is needful for the fact which stares out of these statistics.

Selangor Hospitals, 1891-1893.—*Selangor* is the next largest Western State of the Federation. In the three years 1891 to 1893 (these figures were furnished me by a late colleague then in charge of the District Hospital, Dr. Welch) the admissions to the principal Pauper Hospital at Kuala Lumpur for all causes numbered 9,971, of which Tamils contributed 61, and Chinese 921 per 1,000. The admissions for beri-beri alone numbered 2,601. Of diseases exclusive of beri-beri Tamils contributed 83 per mille, but of cases of beri-beri only 2 per mille—5 actual cases in three years.

In 1899, in the same State, Dr. Lucy, the Acting State Surgeon, informs me the total admissions for all causes numbered 11,420. Of these, 2,332 were Tamils—118 per 1,000. The admissions for beri-beri alone numbered 1,736, or 87 per cent.

Of all diseases, excluding beri-beri, the Tamils contributed 239 per 1,000; but of the cases of beri-beri they contributed only 9 per 1,000 (16 actual cases).

The admissions to hospital for beri-beri annually per 10,000 inhabitants living in this State in 1899 was for Chinese, 132; for Tamils, 10.

Comment can hardly strengthen the significance of these figures. The instances given have not been specially selected from among others. They are the only statistics relative to the Native States which I have been able to obtain.

In the Native States, therefore, the Tamil Immune from Beri-beri.—It is submitted that they prove *the entire practical immunity from beri-beri*, in a highly beri-beric area, *of the Tamil coolie population, the only class among the rice-eating immigrants into that area which is so exempt, the only section of the population which habitually feeds upon cured rice.*

In the Straits Settlements.—The Straits Settlements proper,

although of far smaller area, contained in 1891 some 70 per cent., and in 1900 63 per cent., of all the Tamil population of the region we are dealing with as a whole.

In Singapore and Penang the greater part of all sections of the population is urban, and to a large extent this is so also in Malacca. The latter territory, with Province Wellesley, are the only portions of the Settlements on the mainland, and are contiguous to the Native States, comparison of which can be made more strictly with them than with either of the island urban Settlements, Singapore and Penang.

Tamils somewhat Differently Circumstanced.—Special circumstances make the Tamils, taken as a class, in the Settlements less strictly comparable at all points with their fellows of the same race in the Native States. In the first place, in the States barely a fraction of the Tamil element is permanently settled. Nearly the whole population consists of coolies but recently imported, with, for the most part, a very large preponderance of males. But in the Settlements the population is largely old and settled, one of which the females and children outnumber the males, and which is only in part a coolie or labouring class. Many of the 'Tamils' in the Settlements are born there. Many of them are engaged in trade, a considerable number are clerks in civil employ, etc.

Many better off.—Now, while these conditions, making for exemption from a disease which might be supposed to prevail rather among the lower orders than among those whose standard of living was higher, would, were beri-beri a disorder of infectious nature, undoubtedly tend to secure some amount of protection for the more well-to-do, and so diminish the case-incidence of the epidemic upon the Tamil population as a whole; on the other hand, they would, if the disease is not infectious, and if that view of its nature be correct which has been assumed, rather tend to the result that, among the classes of the population more remote from the coolie level, when the race under consideration is Oriental, the incidence of the disease should be increased. For, not only are the better class among the Tamils necessarily free, and thus not sharers of that wholly beneficial, if quite undesigned, effect which the enforced supply by employers of the customary 'cured' sort of rice for their coolies has had in preserving the latter from beri-beri, but, among all Orientals, and among Tamils especially, the influence of custom and the traditions of caste are less strongly rooted among the highest than the lowest.

Among the latter, accordingly, the use of cured rice is more persistently adhered to ; but the more advanced of their fellows, whom longer contact with strangers has made more catholic, or looser, in their dietetic ways, are obviously more likely to eat, as it is found in experience they do, any variety of rice to which taste or economy inclines them.

The better classes of all nationalities are, in Singapore and Penang and Malacca, treated in the 'General,' the coolie class in the 'Pauper' Hospitals. It is among patients in the former institutions that the largest incidence of beri-beri upon Tamils should be found, if the view just developed be correct. And the returns of the Pauper Hospitals of the Straits Settlements ought, comparably with those of the Native States, to afford the correcter appreciation of the relative incidence upon the coolie population.

I have been enabled to find an analysis of the incidence of beri-beri according to nationality in one only of the medical reports for the Straits Settlements—that, namely, for Malacca in 1897. But the table (p. 194) for the material for which I have already acknowledged my indebtedness to my colleagues, Dr. W. S. Kerr and Dr. J. Leask, gives such an analysis of all cases treated in all the Straits Settlements Hospitals for the three years 1899-1901.

These figures singularly confirm the deduction made above, as to the increased incidence of the disease upon those Tamils who by detachment from the conditions which his conservatism makes specially, if incidentally, protective to the coolie, become more cosmopolitan in dietetic habit, and so more exposed to the dangers which indiscriminate consumption of rice entails.

At Singapore General Hospital.—In the *Singapore General Hospital*, of 1,485 Tamils treated, no fewer than 62 had beri-beri—41 per 1,000—a proportion nearly equalling that of the Chinese in the same hospital at the same time—51 per 1,000—and almost twice as great as that among pauper Tamil patients at the same place and time—viz., 25 per 1,000.

The proportion of beri-berics among the Chinese pauper patients in Singapore was, for the same period, 113 per 1,000.

At the Lock Hospital, Penang.—At only one other hospital in the Settlements was any—and this an exceptionally—high rate of beri-beric incidence upon Tamils noted. At the Lock Hospital, Penang, of 34 Tamil patients, no less than nine had beri-beri.

At Penang Free Population of Indians more Rural and Conservative.—In *Penang Island*, a considerable proportion of the whole population of which is urban, it happens that the Tamils contribute far less to the urban than to the rural section, although forming a far larger proportion of each in Penang than they do in Singapore. As it is essentially the mixing with, and assimilation of, the customs of alien races that tends to increase the liability of the Tamil to beri-beri, so both the conditions obtaining in Penang will tend to lessen the case-incidence of it upon them. For, in so far as the population is rural, it will be preserved by that fact from the imitation of dietetic customs which more intimate association with strangers in towns must induce; and, in so far as the race is, in Penang Town, more numerous relatively to the Chinese than in Singapore, that factor will tend to the conservatism of habits which are racial.

Therefore, also Less Liable to Beri-beri.—The results are as could have been anticipated. The beri-beri rate among free Tamils at Penang is lower than at Singapore. Of 1,109 Tamils treated at the Penang General Hospital during 1899-1901, only 7—or 6 per 1,000—were beri-berics. At the Pauper Hospital, of 3,012 Tamil patients, only 23—or 7 per 1,000—had beri-beri.

But another and more important factor obtains at Penang as distinguished both from Singapore and Malacca. Penang is the headquarters of the local cured-rice industry. It is there that almost all the cured rice supplied to coolies throughout the States and Settlements is produced. That rice is therefore easily obtainable in the shops there, and also more cheaply than elsewhere. In neither Singapore, Malacca, nor the principal villages of the mainland is this sort of rice always procurable.

At Malacca Town—Tamils Addicted to Uncured Rice.—In *Malacca*, Tamils compose less than 2 per cent. of the population, numbering in 1901 only 731 out of a total of 32,293 adult males. Most of these Tamils are coolies living within the town limits, 'old inhabitants,' addicted, Dr. Sheppard, the Colonial Surgeon Resident, informs me, to eating promiscuously any variety of rice obtainable.

There Beri-beri Rate Higher.—In the General Hospital, Malacca Town, 1899-1901, there were treated but 42 Tamil patients—too small a number upon which to base any statistical conclusions. None of them had beri-beri.

In the Malacca Town Pauper Hospital during the same period there were treated 1,006 Tamils, of whom 11 had beri-beri—a

higher proportion than in the same class at Penang, though greatly lower than the corresponding rate for Singapore.

At Malacca padi is largely grown—some is even exported—and rice prepared from the local grain is sold cheaply in the shops, where it finds at least as much favour with the Tamil coolie as imported uncured grain. The local and fresher rice is certainly less injurious than the latter, and its use stands, I think, for an influence at Malacca, which may counteract the increased incidence of beri-beri which forsaking cured rice would otherwise bring upon the Tamil. Beri-beri is severe at Malacca among the Chinese paupers, the case-incidence during 1899-1901 being no less than 143 per 1,000.

Rural Malacca and Province Wellesley.—Coming next to those parts of the Settlements, alone strictly comparable with the Native States, adjacent to which they lie upon the mainland—namely, the *rural* part of Malacca territory, and *Province Wellesley*—the Tamil population of which is entirely of the labouring or coolie class, it will be found that the incidence of beri-beri upon them is even less than that ruling in the Native States. In the two district hospitals of Malacca, Jasin and Alor Gajah, during 1899-1901, 300 Tamils were treated, not one of whom had beri-beri.

Incidence in Province Wellesley.—In the four district hospitals of Province Wellesley during the same period 3,502 Tamils were treated, of whom only 4 had beri-beri—1 per 1,000. Among the Chinese at the same time and place, of 2,692 patients, 163 had beri-beri—60 per 1,000.

Peculiar Freedom from Beri-beri of Province Wellesley—a Padi-exporting Area.—The incidence of the disease is, and so far as records extend has always been, in Province Wellesley, even upon the Chinese, an extraordinarily low one compared with other parts of the Peninsula and the Settlements. It may be interesting to mention here the reason which alone seems likely to account for the peculiar relative freedom of this single but large area from beri-beri. Province Wellesley is, together with a part of Perak adjoining it—Krian—to which similar conditions apply, the only district in which sufficient padi is grown for export in bulk. The whole population is engaged in agriculture, chiefly with this product, and hence it comes about that very little of the uncured Siamese and Rangun rices are imported. The people eat either cured rice made at Penang, closely adjacent, most of it from their own padi, or rice prepared

by themselves from the local grain, freshly husked by hand as required for use.

One more fragment is thus added incidentally to the evidence connecting uncured rice with beri-beri.

Summary of Conclusions from Hospital Statistics.—To sum up, the results afforded by examination of hospital statistics, including no fewer than 128,852 in-patients of Tamil or Chinese race, show :

1. That of all cases treated, beri-beri accounts for 11 per cent.
2. Of all diseases, *exclusive of beri-beri*, Tamils contribute more than one-third—362 per, 1000—Chinese contribute more than one-half—554 per 1,000.
3. Of admissions *for beri-beri only*, the Chinese contribute all but 3 per cent.—876 per 1,000—the Tamils contribute not half of 3 per cent.—12 per 1,000. The balance—1½ per cent.—is contributed by all other nationalities.

Or, to place the same results in other phrase, of all the *Chinese* patients treated, beri-berics were 195 per 1,000 ; of all the *Tamil* patients treated, they formed only 4 per 1,000.

Moreover, relatively insignificant as it is, this rate of incidence of beri-beri upon the free Tamil represents *all the cases* supplied by Tamils of whatever class, and of whatever kind of dietetic habit.

The Exceptions when Examined, all Eaters of Uncured Rice.—It has been mentioned that each of the six cases of beri-beri which have come under the writer's observation proved to be eaters of uncured rice. It has also been shown that the places at which, and the classes among which, cases of beri-beri are more numerous in Tamils are those precisely in which the habit of eating uncured rice is mostly likely to be acquired. Thus, the extremely small total of cases of beri-beri actually observed among free Tamils may reasonably be discounted as being in the highest probability only examples of the disease in Tamils who are eaters of uncured rice.

The Proof thus Completed that Tamils Eating only 'Cured' Rice always Exempt from Beri-beri.—The proposition that so long as he eats cured rice only the Tamil, whether statute immigrant or free, does not incur beri-beri is not invalidated by such exceptions, and may be thus held to be completely established.

The great natural experiment which has thus been in process for a decade and more of feeding two large bodies of individuals, all susceptible to beri-beri, and newly immigrant into a territory,

throughout the whole of which the disease is endemic, upon two distinct sorts of rice, has thus demonstrated more abundantly than any mere laboratory experiment could do the important fact that the incidence of the malady does vary with the sort of rice eaten.

The Deduction thus proved by Natural Experiment that Beri-beri depends on the 'Sort' of Rice Eaten.—No difference of any constant kind, or indeed of any kind conceivably capable of determining the incidence of disease, existed between the two bodies of men dealt with other than the single one, which, on the other hand, was constant, that they ate different sorts of rice—the cured and uncured grain. Throughout the area, during the period dealt with, the result was uniform. Among the eaters of the uncured rice beri-beri prevailed. The eaters of the cured rice were always and everywhere exempt.

But this is not yet all.

Converse of the Experiment also True.—The *converse* of the experiment, so far as it relates to the eaters of cured rice, at least, has also been carried out, and yields evidence which, if less extensive, is almost more valuable, as the attainment of positive results, after the reversal of an experiment which under the first conditions gave only negative ones always must be.

When Fed on Uncured Rice Tamils get Beri-beri equally with Others.—It has been said that the proportion of Tamil patients in Government hospitals generally, in the endemic area, who have beri-beri is an extremely small one, both absolutely and relatively when compared with the Chinese, so that in the Straits Settlements, for example, during the three years only 5 out of every 1,000 Tamils sick had beri-beri, as compared with 107 for the corresponding ratio among Chinese, and that of these cases some were known to be, and probably all were, eaters of uncured rice.

Were individuals of the race, thus exempt from beri-beri on a diet of cured rice, to be fed for a fair length of time—several months—exclusively upon the uncured grain it is to be expected (according to the theory) that the incidence of the disease upon them should become equal, or nearly so, to that upon the ordinary uncured rice-eaters.

This appears to be actually the case, although from the circumstances already detailed, the statistics which can be brought forward to prove the point are not so extensive as those which demonstrate the exemption of the Tamil when, as usually, eating cured rice.

Examples of this among Free Tamils.—The increased incidence of the disease upon those Tamils who are free, their own masters, and by reason of this condition, and because also of longer residence in the country, have acquired to some extent the habit of eating the cheaper and generally more easily procured uncured rice, as compared with the entire exemption from beri-beri of the Statute Indian immigrant coolies, who are strictly fed upon cured grain only, provides a certain amount of evidence in this direction.

In Greater Liability of Urban as compared with Rural, and Upper compared with Lower Classes.—The increased liability of the urban, as compared with the rurally employed coolie, and of the more healthy or educated and less caste-bound classes of the same community, which reaches an exceptional severity (for Tamils) among the clerical and other socially equal sections of this race, represented in Singapore General Hospital, provides more evidence of the same nature. I have already mentioned that the six cases of beri-beri in Tamils, the only ones I have seen during the last eight years, were all eaters of uncured rice.

In Greatly Increased Incidence of the Disease on Tamils in Gaols.—But when it happens that through compulsion a considerable number of Tamils are fed only upon uncured rice for a long time, as occurs when they are incarcerated in a gaol, where the disease is prevalent, and when, as a result, it is found that the ratio in which they become affected with beri-beri in the gaol greatly exceeds that which prevails among them outside, this fact—the converse result of the converse experiment made under precise and controllable conditions—affords final evidence of the truth of the proposition discussed.

Selangor Gaol, 1900.—In the Selangor Annual Medical Report for the year 1900 the acting State surgeon, Dr. Lucy (reporting upon the Pudoeh Gaol), draws attention to the fact as remarkable 'That no less than 16 cases of beri-beri, or 2.93 per cent.—*2 per cent.*—of the total admissions developed among the Malay and Indian population of that institution. Upon comparing this rate with those of other hospitals, we find that for all diseases there were 16,950 admissions, and the cases of beri-beri among the Malays and Indians number 52, or only *3 per 1,000* of the total.'

During the three years 1899-1901 Dr. Travers informs me 385 Tamils were incarcerated in Pudoeh Gaol, of whom 8 got beri-beri. This is a proportion of 20 per 1,000.

172 THE CAUSE AND PREVENTION OF BERI-BERI

In the same gaol during the same period the incidence on Chinese was 90 per 1,000.

During Wright's Experiment 22 per 1,000.—H. Wright,¹ in his observations of beri-beri at Pudoh, states that between May 3 1901, and April 1, 1902, 220 Indians (all but two of whom were Tamils—two being Pathans) passed through the prison. Five of them got beri-beri—22 per 1,000. It is to be remarked that if the proportion of long sentences at this time were the same among Tamils as it was in most years, and among all races, viz., 5 per cent., then only some 10 or 11 of the 'Indians' incarcerated would have been likely subjects for beri-beri, or, in other words, at least half the Tamils who could possibly acquire beri-beri actually did so.

The figures, though too small from which to draw conclusions applicable generally, show at least as a positive fact that the liability of the Tamil to get beri-beri in Pudoh Gaol was relatively greatly increased, not only by comparison with his own liability to the disease when not in prison, but also compared with other races incarcerated in the same prison.

In the Singapore Prison during the great 1878-1884 epidemic the Tamils were, next after Malays, the most severely attacked of all nationalities. Of 671 Tamils incarcerated in three years, 1878-1880, no less than 105 had beri-beri. The incidence on long sentences was 44 per cent. in 1878, and 39 per cent. in 1879. The effect of the uncured rice given them in this case seems to have been accentuated by a relative deficiency of fatty food as compared with the Chinese. In the later epidemic in this gaol they suffered less than the latter race.

In the same prison, during three years, 1899 to 1901, there were admitted to the infirmary 223 Tamils, of whom 10 had beri-beri, a proportion of 45 per 1,000.

The proportion having beri-beri in every 1,000 sick Tamils admitted to the Government pauper hospital outside the gaol in the same settlement, at the same time, was but 24 per 1,000 (see Table, p. 196).

Therefore the incidence of beri-beri upon Tamils in Singapore Prison also was greatly in excess of what it was upon the same race outside.

Much must, in all such institutions, obviously depend upon the relative proportions in which prisoners incarcerated for different lengths of time enter into the totals of each nationality.

¹ Study, p. 51, Table No. 26.

As a matter of fact, in late years, in both the Singapore and Pudooh Prisons, the proportion of long sentences among the Chinese exceeds largely that among the Tamils, so that the incidence of beri-beri upon the Tamils has this factor to reduce it. Were this condition taken carefully into the reckoning, and the incidence upon each race calculated for groups exposed for equal periods, it would, I think, be found that the Tamil who out of prison uniformly escapes beri-beri is, in such institutions, where he is fed on exactly the same food as others, even more sensitive to the influence which causes it than they, and succumbs both earlier and in relatively greater numbers.

At Christmas Island, Durham¹ says, the Tamils suffered severely from beri-beri. They eat uncured rice there.

The Variation of Beri-beri Incidence with Sort of Rice Eaten therefore Concomitant with Variations in Latter.—The incidence of beri-beri upon two races, Tamils and Chinese, immigrant into an area where it prevails, has thus been shown not only to vary, but to *vary concomitantly* with the sort of rice eaten by either class.

Evidence more cogent than this could hardly be required—it could certainly seldom be obtained for any theory; and the proposition asserting beri-beri to depend on rice might very well be left here, be regarded as demonstrated, on these grounds alone.

Much more, however, can, as a matter of fact, be said.

Incidence of the Disease upon the Malays.—Not only the Tamil and Chinese immigrants, but the Malays, the original inhabitants of the region, show variations in the degree to which they are attacked, which bear definite relation to changes in their rice-supply. The differences here apply, however, not to cured and uncured rice, but to *fresh*, as opposed to *stale*, uncured rice.

Malay Population.—The Malays enter into the whole population, in the following proportions, in the different States and Settlements (census 1901):

Male Adults only.				
Singapore	101 per 1,000.
Penang and Dindings	153 "
Province Wellesley	397 "
Malacca	551 "
Perak	248 "
Selangor	137 "
Nagri Sembilan	324 "

¹ *Jour. of Hyg.*, Jan., 1904, p. 119.

174 THE CAUSE AND PREVENTION OF BERI-BERI

The total number of male Malay adults in the whole region in 1901 was 148,755.

They afford, therefore, ample material upon which observation may be made as to the prevalence of beri-beri.

Malays seldom Resort to Hospitals.—Unfortunately, being a shy and diffident race, purely pagan and deeply superstitious (though nominally and ceremonially Mohammedan), as firmly wedded to ancient as averse from adopting new habits, highly domesticated and fond of home and comfort, the Malays (except those in European employ) rarely enter the hospitals. Thus Seremban is the centre of that portion of the Western States most thickly populated by Malays (the population in 1891 was 16,766 male adults), and the inhabitants may be said to be used to Europeans and their ways. Yet during the ten years, 1891 to 1900, while 12,028 patients of all nationalities were admitted to hospital for all causes, but 319 of them, or 26 in 1,000, were Malays (and many of these not natives of the Peninsula, but from islands of the Archipelago, including Javanese, Boyanese, etc.), of whom one-third were police constables.

The Straits Settlements proper have been longer occupied by the British, and the Malays occupying them are consequently more familiar with the ways of the Occident.

Yet analysis of 57,590 cases of all races admitted to different hospitals in the Straits Settlements during three years shows that only 55 per 1,000 were Malays.

Some Vital Statistics of Malay Population.—In 1901, for which year a census enabled fairly accurate statistics to be compiled, the writer reported to the Government at some length on the conditions of disease prevailing among different sections of the population in the Negri Sembilan. In that year it appeared that among the Malays the birth-rate was 40, and the death-rate 29, per 1,000 living annually.

The annual sickness-rate, calculated on the basis that the case-mortality for all races should be the same without as within the hospitals, was 391 per 1,000 annually for Malays, as compared with 416 among Chinese, 1,605 among Tamils.

The admissions to hospital per 1,000 of each nationality living, annually, were :

Europeans and Eurasians	20
Chinese	89
Indians (chiefly Tamils)	395
Malays	2

For a close appreciation of what the prevailing diseases and causes of death among the Malays may be, it is clear, therefore, that hospital statistics alone will afford but little help. Recourse must be had partly to the accounts of the causes of death given by the Malays themselves at their registration of the event, and partly to the impressions and experiences derived by those most conversant with them and their country.

Doubtful if Beri-beri really Endemic at all among Malays.—Seeing that beri-beri is certainly epidemic among the alien population of the Peninsula, it would seem strange should it not be so also among the indigenous inhabitants of the region, the Malays themselves. Yet, as a matter of fact, it has been stated to be doubtful whether beri-beri even occurs, or if now endemic among them whether it has always or long been so.

The Word 'Beri-beri' not Current among Them, nor any Single Name for the Malady.—The word 'beri-beri,' as descriptive of disease, is new to the local vocabulary, and where in use, as among Malays who have become acquainted with the malady and this designation of it by Europeans, is usually pronounced wrongly ('beeri-beeri,' instead of 'berri-berri'). Nor is there any other single word in current use by Malays to describe the disorder, although the vernacular has distinct terms for many other specific maladies, such as small-pox, measles, malaria, epilepsy, asthma, etc. When cases of beri-beri are reported they are described, sometimes in terms of their symptoms, as 'heaviness' (*bērāl*), 'numbness' (*kēbās*), 'tingling,' 'formication' (*sēmūt-sēmūt*—literally 'ants'), 'swollen legs' (*kākī bēngkāk*), or 'paralyzed legs' (*k. lumpoh*), at later periods; sometimes, referring to supposed causation, as cases of being 'touched,' or 'overlooked,' or 'stricken,' by wizard or other malign magic influence (*kena hantu* or *k. bandang*). ..

Now, beri-beri is not a difficult disease to distinguish, more especially in a country where its congeners, arsenical, alcoholic, or other forms of neuritis and myelitis (sources of confusion to the expert) do not occur, and I find in practice that where many cases happen of which they may take notice, the Malays do easily recognise it as a definite and separate complaint. Support is thus afforded to the belief that in whatever degree beri-beri may have existed among Malays during recent years it has not always, nor long done so; has at all events never prevailed to such an extent, nor with such severity, as to become a matter

176 THE CAUSE AND PREVENTION OF BERI-BERI

of popular recognition demanding expression by a particular word in the colloquial.

Doubt has often been expressed as to the occurrence of the disease among the Malays of the Peninsula at the present day, and competent observers have stated that among the Malay population generally it practically does not exist.

Beri-beri Rarely Encountered among Malays.—Two observers, whose position should warrant their evidence being accepted, will be sufficient authority for this statement, and will serve to show the general freedom from beri-beri of the Malay, as he lives naturally in his own villages.

At a conference of medical officers of the Federated Malay States held at Kuala Lumpur in 1898, Fox, Acting State Surgeon of Perak, whose service in that State for many years had given him ample opportunity for forming an opinion, said (in a discussion introduced by the writer as to the relative race-incidence of beri-beri) that he considered the disease to be *as rare among the Malays as among Tamils*—among whom, it had been then stated, it practically never occurred.

H. Wright, in his 'Study' of beri-beri published from the 'Institute of Medical Research' (Vol. II., No. 1, 1902, p. 14), says (par. 113): 'Careful inquiries amongst the Government medical officers, and personal observations all over the Peninsula, lead me to believe that it [beri-beri] is a rare affection [among Malays].' In par. 116 this observer goes on to say that he had 'been in every native village of importance along the west coast of the Peninsula and in the interior. Careful inquiries have failed to elicit, except rarely, the history or any evidence of the presence of beri-beri [among Malays].'

Again, par. 214 *et seq.* :

'My own experience of the Malay Kampongs is that beri-beri is almost unknown in them. The farther the Malay population is removed from centres of civilization the less beri-beri is seen in it.'

'In Kampongs, near Kuala Lumpur, I have seen several cases of severe beri-beri in both men and women. But the men have been *carters* who have travelled about the country *working for Chinese*, and in frequent association with acute beri-berics in well-known beri-beri foci.'

'My own observations are confirmed by others. Mr. Nelson Annandale, who spent nine months during 1901-1902 in Upper Perak and the Siamese States on anthropological work, saw but

two cases amongst several thousands of natives whose whole diet consists of locally grown rice and locally cured salt-fish.'

I have underlined four words in this extract, which are greatly significant to those acquainted with the facts. It happens to be an established custom of the country that Malays, especially carters, engaged in permanent employ by Chinese, should receive from the latter over and above the cash wages a regular and usually large allowance of *rice*, which is of course assigned out of the towkay's own store, and is of the uncured and stale variety. The 'association with acute beri-berics in well-known beri-beri foci,' so conveniently assumed to explain the exceptional case of these Malays, is thus not required to explain the result.

Yet the Malay is very susceptible of beri-beri. The case of the Malay crews of the Pearling fleet, quoted from Haynes above (p. 61), affords one proof. The case of the Filipinos (native of the Malay stock), who in the great epidemic at Manila in 1882-1883 were¹ *the only race* attacked out of many exposed,² is another. The ravages caused by it among the native troops of Malayan stock employed by the Dutch in Sumatra, both in their army and their fleet, are notorious. In the Peninsula, H. Wright himself, in a later paragraph of the 'Study' already quoted (par. 340), has observed their susceptibility to the disease when in gaol, noting it to be even greater than that of other natives confined with them at the same time and place. He showed that in a party of mixed nationalities under his observation, among whom beri-beri developed, the incidence on Malays was greater than on any other nationality, 8 cases developing among 94 persons during 11 months, or 8.5 per cent., as compared with 7 per cent. among Chinese, and 2.27 per cent. among Indians.

This observation concurs with that made in the Singapore Gaol epidemic of 1878-1884, when, however, the proportion of Malays attacked was absolutely very large, and relatively to other prisoners enormous. Then, from two-thirds to three-quarters of all the Malay long sentences, and one-third of the total Malays incarcerated were attacked—228 out of 747 Malay prisoners had beri-beri in the three years 1878-1880. In both the increased incidence has been explained as due to excess of uncured rice in the dietary.

¹ And still are. The Scouts (Filipinos) contribute the only cases in the American Army of occupation in the Philippines to-day.

² See pp. 99, 223.

178 THE CAUSE AND PREVENTION OF BERI-BERI

In Hospitals Malays six Times more Liable than Tamils.—Moreover, the collection of statistics from hospitals of the Native States and Straits Settlements for several years, presented in this section, shows that beri-beri is among Malays, if not as common as in other races, yet a fairly frequent disorder. Of 4,748 admissions of Malays to various hospitals, 144, or 30 per 1,000 were for beri-beri—six times the incidence on Tamils.

We have therefore, to reconcile two seemingly conflicting observations. The record of quite a large proportion of undoubted cases of beri-beri among Malays treated in hospitals, and a widespread belief among competent authorities that Malays are only with great rarity attacked.

The Writer's Observations.—The writer's own observations, made during more than seventeen years' medical charge in the Malay States, and based upon a fairly close as well as extended knowledge of the country and people, enables him to confirm and to reconcile both these statements.

Under Primitive Conditions the Malay never gets Beri-beri.—It is true that among Malays at large, the population regarded as a whole, beri-beri is rare. I may even venture to go further and say that *among these natives, so long as they lead their primitive pastoral and agricultural life*, untouched by the influences which march with a civilization represented by encroaching hordes of Chinese, *beri-beri never occurs*. On the other hand, absolutely a very large, and, as I think, an increasing number of cases of the disease does annually happen among them under altered conditions.

They are Unusually Susceptible to It.—There is evidence that Malays are even more sensitive than are some other nationalities to the influence which causes beri-beri, so that, where the special circumstances which induce the disease exist, it severely affects them, but where those conditions do not apply, and they do not apply to the bulk of the Malay population, the disease is, on the contrary, rare.

What are the conditions, and what is the essential one, necessary for the acquisition of beri-beri by the Malay? What condition is there capable of determining disease-incidence on the few which does not pertain to the mass of the population of this race? Correctly to answer this question is, of course, to designate if not the immediate cause of the disease, at least the means through which it is incurred.

It is among Malays living at home in their own 'kampongs'

(or village homesteads), tilling their own fields, growing their own *padi*, and *subsisting upon fresh rice* prepared from it daily by themselves, that beri-beri is rare, or even never seen.

Among those of them who have, in search for more profitable employment, or through force of other circumstances, become divorced from these associations, and who, as a result, among other changes have to use for their food rice differing from that to which they have been accustomed, beri-beri is, on the other hand, common, and often unusually fatal.

Malay Characteristics make them seek Labour away from Home.—Malays are at no time fond of strenuous task; it revolts their pride to be seen employed in any drudgery of toil near home. Most of the tilling of their land and the care of their beasts is left to their womenfolk. The men, when they must seek employment, generally leave their own villages, and go further afield, mining, or seeking jungle produce, undertaking contracts in felling and earthwork. Or they engage on ships, in domestic service, or in offices for a few years—a ‘wanderjahr,’ the object of which is usually the accumulation of a small sum of money to get married on, or for some other purpose, and when the specific sum required is obtained they return to their own villages to live in comfortable idleness as long as possible after.

Then they Acquire Beri-beri.—It is while abroad upon such expeditions, whether in small bands searching for jungle produce (rubber, rotans, etc.), or mining in the hills for tin, as sailors or pearl-divers, as tin-washers and ditch-watchers on hydraulic mines—tasks which keep them employed and away from home—camping out, as it were, for several months or more, that Malays are peculiarly apt to get beri-beri. To these employments must be added the men who engage in even larger numbers as police constables, whom the exigence of their service compels to be absent for even longer periods from their homes.

Instances Numerous.—Numerous cases could be adduced to exemplify the liability of the Malay to get beri-beri under the conditions described. The following are such examples:

1. Epidemy at the Paroë Mine.—In 1896 a large hydraulic mining enterprise was opened under European management in a valley called Paroë, near Seremban. Occupation was given to Malay men and women at this mine, a daily average of 80 to 120 of them being employed. Their work was of various kinds—washing tin out of the tail-races or in the washing-shed, clearing

ground and tracks, and cleaning and repairing the ditch and pipeline through which the water-power for the mine was brought from a distance of several miles away in the mountains, the ditch itself running at an altitude of some 2,000 feet.

The quarters occupied by these Malays were especially built for them. The site was old grass land, high and dry and unusually well ventilated, on a spot where there had been no previous occupants. Nearly all the coolies were brought from another State—Perak, from villages which, equally with their new location, were free from beri-beri. None of them had had the disease before. Most of them were ignorant of its name, and unacquainted with its nature.

Some Chinese were also employed at the mine, but they lived apart from the Malays, and came into contact with them only casually in the course of work. There was (until long after its occurrence among the Malays) no beri-beri among these Chinese. Nor was the disease prevalent in the neighbouring Malay villages, with the inhabitants of which those at the mine naturally had association.

Within some six months of the arrival of the Malays at this mine beri-beri broke out among them. Cases occurred day after day, most of them, at least at first, among those coolies who were working on the hill (ditch) section, where the climate was, for them, depressing, being as compared with the lowland misty, raw, and cold.

Such conditions, in the writer's experience, have a marked influence in determining the appearance of symptoms in patients who would otherwise not complain of any. The subjects of beri-beric relapses have over and over again assured him that wetter or colder weather at once recalls or aggravates the cramps and nervous troubles peculiar to the disease, much as the east wind tries the rheumatic or gouty at home.

In this manner mild degrees of the disorder are readily betrayed or, on the other hand, masked by slight changes in the surroundings of the subject, such as damp floors, etc., which should not be (though they often are, however) for that reason regarded as their cause.

Later, coolies on all sections of the work became affected. The symptoms, although sometimes ending fatally, were never pronounced. Often no outward signs would be noted, and but scanty indications obtained even by a medical observer, of the definite presence of the disease. The first subjective symptoms described would usually be a sort of lethargy—a feeling of general lassitude and indisposition to toil. Such a condition, of course,

could easily be mistaken for mere moral indolence by the unsympathetic or inexperienced. Loss of appetite, a sense of repletion, even pain, after meals would follow, and muscular tenderness. Then œdema, numbness, tinglings, formication and paresis, aphonia, cardiac crises, and all the usual train of symptoms, following on in one case or another, left no doubt as to the nature of the malady prevailing among the coolies.

First and last some 1,500 different Malay coolies were brought in to labour on this mine—where the disease still prevails¹—and, I believe, if not all, nearly all the males sooner or later became affected in some degree. As it was found difficult, if not impossible, to persuade the natives to substitute cured for the uncured rice which they were using, the plan was adopted of removing every coolie, as soon as he showed the slightest indisposition indicative of the disease, to the sea-coast, and thence, if improvement did not set in early, to his own home. A few weeks of the change usually sufficed to remove all signs of the disorder, and most of those who had been thus attacked returned of their own accord to the mine, where they as often as not acquired the disease again.

Females, here as elsewhere, afforded fewer patients, but among them also several cases occurred. There were many children, but none became obviously affected.²

These coolies were paid high wages, and had an ample and varied dietary. They bought their own provisions in the shops. *They all ate stale uncured rice.*

2. Epidemy at the Malacca Training College.—A training-school for Malay teachers was instituted at Malacca on March 1, 1900. Some fifty to sixty youths, aged from sixteen to twenty, selected from different districts in the various States of the Malay Federation and the Straits Settlements, were brought together there. The quarters occupied consisted of a large masonry building, formerly the residence of a British official, and subsidiary buildings, on the native model, newly erected. There was plenty of accommodation—no overcrowding.

The course of study was from two years to four.

The site and surrounding houses were, as far as was known, perfectly healthy. The actual location was on a sandy 'lande'

¹ September, 1902.

² I believe that in children cases occur as often as in adults, but that as the symptoms slightly differ they are unnoticed when mild, or wrongly interpreted when severe, and so prove fatal before any œdema or paralysis is produced. They thus go unrecognised.

upon the very edge of the sea. None of the boys had had beri-beri, and none of them came from homes in which there was beri-beri. Although the disease is endemic in Malacca, it is not so among the Malay section of the population.

The dietary was liberal, comprising all such things as Malays customarily eat. The rice used, the best to be bought in the shops, was uncured Siamese or Rangoon.

The circumstances of these boys therefore were in all respects unusually sanitary. They were well-housed, well-fed, amply provided with physical amusement and recreation, and lived in a sea-breeze.

Yet a few months after the establishment of the school it was observed that the inmates were not enjoying good health. There were complaints, on the part of many of the boys, of lassitude and fatigue, disinclination to work, and of more pronounced signs, such as pains about the limbs, stiffness on exertion, and particularly heaviness and weariness in the legs (*kebas*), and pain and cramps in the calves. Some of them developed swelling on the shins. Medical advice was sought, and it was suggested at first that the ailment was something of the nature of rheumatism, and due possibly to the pupils sitting while at school-work over a cement floor. This was accordingly replaced by a wooden floor. But no improvement took place in the general health of the school.

The disease was worse in the wetter months, and its recurrence every autumn with increasing severity led the authorities to consider whether it might not be necessary, in the interests of the health of the pupils, to remove the establishment to another locality, or disband the school.

In 1902 the writer visited the school, found distinct beri-beri in some of the pupils, and came to the conclusion that the illness prevailing was, and had been, of that nature in all the cases.

It was advised that the boys certainly affected should be dieted entirely upon cured (Penang) instead of uncured (Rangoon, or Siamese) rice, and that, for an experimental demonstration of the share played by the latter varieties of rice (different from the kind—fresh rice—to which they had been accustomed at home) in producing the epidemic among the boys, one half of them should thenceforth be given cured instead of uncured rice in the daily rations, and the result as to health be carefully recorded.

The dislike of the boys to the Penang rice (which, although

cured, was ill-cured, and had a disagreeable sour odour) made the headmaster unwilling to insist upon the experiment being so carried out in entirety. All the beri-beric boys were fed on cured rice, however, and they recovered, while of those who persisted upon the uncured variety one got beri-beri, and was sent home by the Colonial Surgeon.

The headmaster, persuaded by a demonstration which might not, perhaps, have convinced those more sceptical, determined afterwards to feed the whole school upon cured rice only. The session during which this change was adopted was marked by the entire absence of cases of 'kebas'—the first term to be so free—and the school has remained free of all signs of beri-beri since that date.

For the following further details as to beri-beri at this school I am indebted to Mr. F. Robinson, the headmaster in charge in 1903:¹

'The first outbreak began with the sudden death of a student from the disease on the morning of November 21, 1900. In the following days four more boys were found to be suffering from beri-beri, and it was deemed advisable to close the college as soon as possible. By November 30 all the boys had gone home, and during their absence the place was thoroughly fumigated and disinfected.

'The boys were away for two months (till February 1, 1901), but shortly after the reopening of the college two rather doubtful cases of beri-beri were reported. They were evidently very slight, for though the boys were sent home they soon returned and were certified by Dr. Croucher (the Colonial Surgeon) as quite well.

'Again in the autumn, shortly before the college was closed for the holidays, there were two more cases, one of the boys being sent to the hospital, and one home, while nearly half of the boys complained of "kebas" about this time.

'After the holidays there were no more complaints until about the spring of 1902, when four boys were sent home suffering from neuritis.'

With regard to this last outbreak I cannot do better than quote the report of Mr. Lornie, who was at the time in charge of the College. Mr. Lornie says:

'About the beginning of May the old complaint of "kebas"

¹ Who has also been good enough to read and vouch for the accuracy as to facts of the above account.

began to be heard, and several boys went home for short periods on account of it.

'By the advice of the Colonial Surgeon "parboiled"—i.e., Penang "cured"—rice was given on alternate days, and as Dr. Braddon, of Seremban, who visited the college at the end of May, pronounced one of the "kebas" patients to be suffering from incipient beri-beri it was decided that the parboiled rice should be given every day to all who had suffered from "kebas," and to any others who preferred it. Twelve boys in all took the parboiled rice, but the majority preferred to stick to "ordinary" rice.

'During the next two months the number of sufferers from "kebas" gradually diminished, and there were no more complaints from those on ordinary rice.

'On July 20, however, a Selangor boy, who had been suffering for some days from "itch,"¹ was pronounced by the Colonial Surgeon to be suffering from beri-beri, and was sent to his home. He had been on ordinary rice, and in consequence all the boys were put on parboiled rice, and as this proved very satisfactory nothing else was given for the remainder of the year. I may add that the ordinary rice has never been reverted to, only the parboiled being used now (August, 1903).

'Of the original twelve, only three boys never admitted that they were thoroughly cured, although they admitted that they were much better, and nothing more was ever heard of "kebas" among any of the other students.

'Since the outbreak spoken of, beri-beri has been entirely unknown among the boys, who have apparently quite got over their aversion to the "parboiled" rice.'

3. **Hose's Experience among Dyaks and his own Case.**—Hose, of Sarawak, in an important paper upon the etiology of beri-beri,² which seems not to have gained the publicity which is its due, mentions the fact that among Dyaks (natives of Borneo) beri-beri is almost always incurred while they are away upon jungle excursions, when the rice eaten is not the freshly-husked grain to which they are used, but shop rice, often become mouldy during travelling. Hose himself acquired the disease while upon such a journey. A valuable experiment by the same observer

¹ Purely subjective itching—general pruritus without any skin lesion—is an extremely common initial symptom in beri-beri, as it is in 'kriebelkrankheit' (ergotism).

² Reference: *Medical Review*, June, 1903. Also *British Medical Journal*, October 28, 1905, p. 1098.

as to the relative toxicity of such stale as compared with freshly-husked rice for food will be cited later on (see p. 308).

4. **Cases in the Writer's own Practice.**—The writer has in his own experience met with many such cases of beri-beri in Malays who have acquired it for the first time during prolonged absences from home, or upon expeditions into the jungle.

5. **Mr. Skeat's Case.**—Mr. W. Skeat, the leader of a Cambridge scientific expedition into the Malay States, affords another such instance of a European who got the disease. While upon this journey his staple food was uncured rice of the same kind as was eaten by his coolies, some of whom also got beri-beri.

6. **Travers—Malay Police at Kuala Lumpur.**—Examples of beri-beri among Malay *police* are numerous.

Travers, in the annual medical report to the Government of Selangor for 1897, narrates an outbreak among the Malays of the local force at Kuala Lumpur. Among the police there were 399 admissions to hospital for all causes during the year, seventeen for beri-beri. 'Fifteen of the cases of beri-beri occurred among the [Malay] police stationed at the central police station. This seems to point to some local cause as the origin of the disease.'

7. **Police in Negri Sembilan.**—In Negri Sembilan, beri-beri is common among the police. Cases occur from time to time at nearly every station. During the ten years 1891-1900 I find that 102 Malay constables were admitted to the Seremban Hospital, of whom 10 had beri-beri. The number of free Malays admitted during the same period was 198, of whom only 8 had beri-beri.

The incidence upon police admitted to hospital was thus double that upon independent men, although relatively few of the cases of sickness of any kind among them are seen, as they prefer to go to their own homes when sick.

In 1901, out of a force of 219 Malay police in the Negri Sembilan, 3 died of beri-beri who were never admitted to hospital.

In February, 1902, I examined 19 Malay police in barracks at Seremban, being that part of the local contingent, 50 in all, off duty at the moment. Seventeen of these men showed definite signs of beri-beri, by which later 2 became seriously affected, and from which a third died.

Five others of the same force were subsequently admitted to hospital.

8. **The Unmarried Men most Affected.**—The barracks—new ones—in which these men were living were only a stone's throw from the gaol, and from the Sikh police barracks, where beri-beri had

never been prevalent. The greater number of the cases were men who had come in but recently from out-stations. There were two features common to all the patients—namely, that the rice habitually used by them was uncured Siamese, and that they were all either unmarried, or men whose wives had not been living with them for some time.¹ This fact their Sergeant-Major connected with the disease in this manner: Those who had wives with them, I was assured, used Rangoon rice as this was cheaper and economy was, with a family, a necessity. The bachelors, indifferent to economy, ate Siamese rice, which, if dearer, was more palatable, either cooking for themselves, or more often feeding at the 'punch-houses,' where the same sort was served, and in both cases probably actually eating *more*, for their rations had not to be shared with a family.

This seemed to point to a difference as to beri-beri production between the varieties, Siamese and Rangoon, a possibility for which there is, in my experience, other evidence. I advised all these police patients at once to change to 'Kling' rice, and to use no other. None of them did so, because they found it too dear, and also unpalatable. They adopted Rangoon rice all round, however, and certainly, since that change, fewer cases have developed among them. The Sergeant-Major, himself a patient, adopted the Penang rice with, as he tells me, immediate and persisting benefit. The active signs of the disease (swelling, cramps, weakness and sensory nerve disturbances), rapidly disappeared, and have not since recurred.

9. **Epidemy at Ampangan—Increase in Villages where Padi Culture is Replaced by Mines.**—During the last two years there has been quite an epidemy of beri-beri at Ampangan, a village near Seremban, with many deaths, an invasion which the natives declare never to have happened before. They themselves attribute it to the fact that during the last two years, their padi-fields having been expropriated for mining purposes, they have had to feed on shop 'Siamese or Rangoon' rice.

10. **Hospital Statistics—More Beri-beri in Towns.**—The hospital statistics, although including relatively few Malays (45 in every 1,000 cases treated) show clearly that their liability to get beri-beri is greater when living in towns than when in the country.

Singapore—57 to 82 per 1,000.—Thus, at Singapore, the Malay population of which is entirely urban, the rates of incidence were

¹ Compare with this the same interesting condition noted by Laurent in the Chantabun epidemy (p. 258).

57 cases of beri-beri per 1,000 Malays admitted at the General, and 82 per 1,000 at the Pauper Hospital.

Penang Town, 17 per 1,000 ; Penang Rural District, nil.—At Penang at the Town Hospital and the Pauper Hospital,¹ the rate was 17 per 1,000, as against a nil record for the country station (Balik Pulau).

Malacca Town, 36 per 1,000 ; Malacca Rural, nil.—At Malacca the incidence in the town area was 36 per 1,000 at the General Hospital, 18 per 1,000 at the Pauper Hospital, as opposed again to a nil record in the country hospitals.

The singular fact is thus brought out in the table of relative racial incidence that in the purely country districts of the Colony no Malays at all were admitted during three years for beri-beri, even at district hospitals, where the disease caused numerous admissions among Chinese, and where the relative number of Malays treated for other ailments showed that they were not averse to seeking hospital aid.

Province Wellesley, nil.—The hospitals were those of Butterworth, Bukit Mertajam, and Sungei Bakap in Province Wellesley ; and Jasin and Alor Gajah in Malacca. Six hundred and twenty-seven Malays were in-patients at these hospitals, among whom there was not a single case of beri-beri.

The districts included the whole of Province Wellesley, and the rural part of Malacca territory. They are also, it is significant, the only districts in which padi is produced locally in quantities exceeding local demands for consumption.

Negri Sembilan.—In the Native States similar conditions obtain. In Negri Sembilan, for instance, of the eighteen cases treated, all but four came from the headquarters station, Seremban, which is not now the centre of an agricultural district ; so that the Malays there live chiefly on imported rice.

Pahang.—In Pahang, the incidence was greatest, out of four stations, at *Raub* (45 per 1,000). Raub is a populous mining village remote from agricultural areas, at which all the Malays employed feed on imported rice. At *Bentong*, in the same State, another mining centre, but placed in the midst of a padi-growing settlement, and long and still notorious as one of the worst 'foci' of beri-beri, where from year to year half or more of the Chinese employed get it, the Malays have never suffered. They grow padi sufficient for their own consumption.

II. Prisons.—In the prisons the relative liability of the Malay

¹ See pp. 194, 195.

to beri-beri is much greater than it is outside. I have quoted Lucy as commenting on this fact in Selangor in 1899. I have also cited from H. Wright the observation made later in the same prison that the Malay proved even more susceptible of the disease than prisoners of other nationalities.

In the Singapore Prison, during the three years 1899-1901, of 233 Malays admitted to the Prison Infirmary, 21 had beri-beri, or 88 per 1,000, a higher rate than was exhibited at any of the hospitals, the statistics of which are given in the table of Relative Incidence.

In the Singapore grave epidemic of 1878-1884, when all natives suffered badly, the Malays fared far the worst. It is unnecessary here to repeat the circumstances, which have already been detailed, but the facts may be recalled that at that time the rice eaten by them was uncured; that it was eaten in excess by all the prisoners (28 ounces per diem), and relatively in greatest excess by the Malays, upon whom the incidence of beri-beri was correspondingly most severe.

12. Sporadic Cases in the Writer's Experience.—Of examples of beri-beri occurring *sporadically* among Malays the writer could cite many coming within his own experience. They are mostly young men in civil employ, in offices as clerks, draughtsmen, etc., and in the fields as forest rangers and surveyors. There is never any factor in common which could account for the incidence of the disease except the one, which is invariable, that they no longer live in the primitive fashion of the Malay in his own home; they no longer subsist on fresh rice.

13. Epidemic at Port Dickson Estate.—It is not often that a difference in the incidence of beri-beri is noted among Malays living and working together at one and the same time and place. But of such a difference, exhibited both by Malays and several other nationalities, the following is a conspicuous instance:

A large coffee estate was opened up near Port Dickson in the year 1894. A great many coolies of all kinds were employed at the different operations of felling, clearing, holing, planting, weeding, building, etc., and first and last several thousand individuals came on the labour roll.

Those employed were: (1) From 50 to 60 Malays of the immediately surrounding villages, and others from more distant parts of the country; (2) from 30 to 40 Javanese working and temporarily domiciled in, but not belonging to, the country; (3) from 60 to 80 Chinese, both old hands and sinkhehs; (4) 80 *Ban-*

joerese, natives of Malay race, imported from Southern Borneo ; (5) from 200 to 300 Tamils, natives of Southern India ; (6) 3 or 4 Bengalis employed as watchmen and shop-keepers. There were also some Eurasians and Europeans.

The quarters provided for the coolies were roomy, well-ventilated dwellings, with plank floors, and sleeping benches high off the ground ; there was no overcrowding ; the sites were high and well drained ; the water-supply was from excellent deep wells. All the hands were well paid, and able to buy ordinary provisions of good quality, at lower than market prices, at the estate shop. The whole area of the estate had previously been uninhabited jungle ; the houses, entirely new, could have received no possible heritage of infection from previous occupants.

Yet beri-beri prevailed upon this estate almost from the beginning, and speedily assumed such proportions as seriously to hamper the management. The first cases occurred among Chinese, many of whom, chiefly sinkhehs, were affected. Some of the Javanese also suffered, and some of the Malays who came from a distance. But the chief victims were the Banjoerese. In the village from which these people came, they stated, beri-beri was unknown, so that the disease was for them a calamity of a kind not previously experienced. Throughout the voyage, of about twenty days, they had been unaffected ; they had arrived in good health at the estate, and remained so there for about two months after their arrival. The disease then began ; both men and women were severely affected ; many died ; and shortly such a proportion of them had succumbed that the only way of saving the remainder seemed to be to send them back to Borneo. None of the Malays belonging to the neighbourhood suffered, nor was beri-beri prevalent in any of their villages. The Tamils, although very sickly—suffering especially from dysentery, diarrhœa, and fevers—remained, as usual, absolutely untouched by beri-beri, as were also the Bengalis, the Eurasian assistants, and the European managers of the estate.

The rice supplied on the estate was of the two common sorts—cured (Penang parboiled) and uncured Rangoon or Siamese. The Chinese and Javanese, the Malays who had no village-supplies of their own, and the Banjoerese after arrival, all of whom furnished cases of beri-beri, ate uncured rice. The Banjoerese before their arrival had subsisted on freshly-husked more or less red rice of their own making, as did the Malays of the vicinity, neither of them getting beri-beri under these circumstances.

The Tamils, and also the Eurasian dressers and assistants, were uniformly fed on cured rice, and the Bengalis and Europeans ate no rice at all.

14. **General Exemption of Malays in Villages where Chinese when Introduced get Beri-beri.**—The instances of beri-beri among Chinese following their intrusion into Malay villages, the inhabitants of which have themselves previously been, and continue to remain entirely free from it, have already been referred to early in this work. This sequence of events, noticed almost everywhere throughout the peninsula, renders the conclusion irresistible, that the influence which one section of the population thus continually incurs, and the other (in innumerable instances) as uniformly escapes, can be nothing derivable from a factor common to both—cannot, for instance, be determined by the locality in which both dwell.

In all such cases it is invariable that the usual difference in the sort of rice eaten by the two races obtains, the Chinese using uncured, the Malays fresh rice.

The answer to the question then, What factor, capable of determining disease, is there, absent from the circumstances of the great mass of the Malay population, who, living in primitive fashion in their villages, escape beri-beri, but common both to all those Malays who forsake these conditions, and to the aliens—whether in their own or other countries—who get beri-beri? must have become increasingly evident from the facts which have been considered.

The Reason of the Exemption of the Malay under Primitive Conditions.—In his undisturbed home the staple food of the Malay is fresh rice, grown, reaped, and husked by himself. The small portion taken from the store for daily use is, when husked and winnowed, sorted over with the greatest care, and picked free of all unsound grains. Dirt is removed by copiously washing the rice before it is cooked.¹

At Home he eats always Fresh Rice.—Rice is thus prepared by

¹ If, as a fact, free Malays, even when dieted on uncured rice, contribute a ratio of cases proportionately smaller than the Chinese—a point which wide enough statistics have not been collected to make evident—a reason for mitigated prevalence of the disorder among them might be found in the fact that the habit of hand-picking the grain and thoroughly rinsing it in cold water before cooking is still adhered to by them whatever the grain used, and under whatever circumstances it may be eaten, provided, that is, there are women to perform these functions. These details of culinary care are generally neglected by the Chinese.

the householder only for family consumption. It is seldom made a commodity of sale. Only in one or two areas in the whole of this region is the quantity of padi grown more than is sufficient for local needs. In these places small parcels of the locally-produced padi are bought by the Chinese, who husk and sell it. But then it is exposed for some time after husking and becomes stale. In none of the villages, except perhaps Malacca, is any considerable quantity of such *quasi-fresh* rice to be found at any time on sale.

Abroad he is unable to obtain Fresh Rice.—Hence it comes about that, when the Malay leaves his home, whether to take part in a jungle excursion, which may be weeks or months in duration, to join a ship, to go pearl-diving, to work at a distant mine, or to engage in police or civil employ, it becomes a part of his changed conditions, that the kind of rice—fresh rice—to which he has always been accustomed, can no longer be obtained.

And therefore uses the Cheapest Uncured.—Under these circumstances he seeks as a substitute the cheapest rice resembling it which the shops at hand supply. Almost invariably this is stale, uncured Rangoon or Siamese rice. The former is usually a cent or two cheaper per *gantong* (gallon measure), but is less esteemed for flavour than the Siamese.

Thus he becomes as liable to Beri-beri as all other users of Uncured Rice.—There has been shown some reason to believe that the former of these sorts of uncured rice is less often, or perhaps to a less extent, poisonous than the latter; but, however this may be, it being upon one or another of these two kinds that all those classes of people feed who are most wont to get beri-beri, the Malay using them ranges himself thereby under conditions which are in all important respects the same as those applying to Chinese. It is not surprising that as a result he should acquire the disease.

This Difference in Rice used the only one between Malays in their Villages and Malays Abroad.—This distinction in the sort of rice consumed is the only difference (conceivably capable of causing disease) which can be asserted to exist between Malays who, dwelling in their own villages, subsist upon fresh rice and escape beri-beri, and Malays who, under all other kinds of circumstances between which nothing else can be shown in common, except this change of food, acquire the disease.

The Dyaks studied by Hose, the Malay divers, of whose sickness in the pearling fleets Haynes gives account, the boys in

the Malacca school, the Banjoerese coolies on the coffee estate, the Malay police all over the Federated States, the Malay prisoners affected in the gaols, had this one circumstance only common to them all—that they were being fed upon uncured instead of the fresh rice to which they had been accustomed, and which the experience of ages shows to have been, and still to be, innocuous.

Wright (H.), it is true, referring to the matter of rice and beri-beri as affecting the Malay, implies, if he does not directly state, that in the Pudoah Gaol epidemic in Selangor, in 1901-1902, which he was studying, Malays got the disease independently of the consumption of rice. He says ('Study,' par. 319): 'It cannot be assumed that Malays as a body escape beri-beri because of the absence of some dietetic factor that obtains with the susceptible Chinese, for a personal observation in the Kuala Lumpur Gaol elicited the fact that on their own chosen diet, cooked by one of themselves, they developed beri-beri in a larger proportional percentage than either the Chinese or Tamils.'

The suggestion here is, that the Malays had some option about their rations; that, exercising it, they selected food different from that taken by the Chinese and Tamils; and that, although thus feeding upon a different diet, they yet acquired beri-beri. But reference to par. 340 of Wright's own Report shows that the Malays referred to belonged to 'party No. 2'; that party No. 2 was one of several into which the prisoners were divided for observation from May 3, 1901, to January 1, 1902; and that *between these two dates all the prisoners in the gaol had a diet identical in all respects, save only as regards the sort of meat eaten*. The 'choice' of the Malays, therefore, amounted to no more than the fact that they were, in deference to their religious prejudices, allowed buffalo instead of pig-meat. But each Malay, in common with every other prisoner in this gaol, received daily during the period reviewed by Wright no less than 21 tahils—i.e., 28 ounces—of uncured rice (weighed dry) for his daily ration. In par. 286 of this Report Wright says: 'The rice was a good quality of Siam or (*sic*) Rangoon.'

This 'personal observation' of Wright's, so far, therefore, from making it impossible to assume 'that Malays as a body escape beri-beri because of the absence of some dietetic factor that obtains with the susceptible Chinese,' supports such an assumption, on the contrary, very actively, and for that reason I quote it.

Conclusion certain : that in Malays the Incidence of Beri-beri depends on sort of Rice Consumed.—The evidence afforded by a comparison of the circumstances of the Malays who do, with those of them who do not acquire beri-beri brings this race, therefore, into a line with other nationalities. *When feeding upon fresh rice they escape ; when they eat uncured rice they get beri-beri.*

The disease in them depends directly on the sort of rice consumed.

The table following gives the statistics of incidence of beri-beri on different nationalities which have been cited in the text (see pp. 194 and 195).

Malays of the Archipelago.—Not only in the States of Malaya under British protection, but in all other countries of the Malay region where the inhabitants use rice, conditions prevail similar to those depicted. Everywhere it is the custom of the Malay dwelling in his own homestead to preserve his rice in the husk, guarding it from wet especially with the greatest care. From this store so much padi is taken from time to time as will suffice for a few days' consumption. The rice required for daily use is made fresh every day from this padi. I have learnt from natives themselves that this is the custom in Java, Sumatra, in Borneo, and the Celebes. In all these countries it has been observed—by Rupert¹ in South Borneo, by Van Leent² in Banka, New Guinea and Sumatra, by Vorderman,³ Laoh,⁴ and others in Java and Madoera—that the natives living free in their villages never, or but rarely, suffer from beri-beri, even at times and places when new-comers among them do so.

More than this : the perplexing fact was also long observed by them that so soon as the natives of any one of these districts went into European service, as soldiers, sailors, or police, where they got Government rations, or entered gaol, they at once suffered from beri-beri. This happened (it seemed) just the same even when the diet given them was purposely made (so far as the observers understood its details) the same as that to which they had always been accustomed. Rupert, noting these facts, had all his men who kept getting beri-beri at a South Borneo outpost put exactly (as he states) under conditions in every respect the

¹ *D. arch. f. Klin. Med.*, xxvii., p. 499.

² *Gen. Tijdschr. v. Ned.-Ind.* (N.S. ix.), p. 272.

³ 'Onderzoek,' 1895.

⁴ 'Iets over die Ætiologie, etc., der Beri-beri,' 1903, p. 89.

TABLE SHOWING RELATIVE RACIAL INCIDENCE OF BERI-BERI AT VARIOUS GOVERNMENT HOSPITALS FOR SEVERAL YEARS AMONG THE PRINCIPAL DIFFERENT RACES OCCUPYING BRITISH MALAYA.

The Admissions are Classified according to the Nature of their Staple Diet.

In each column (a) = total cases admitted for all diseases, (b) = total cases of beri-beri admitted, (c) = case-incidence of beri-beri (proportion of cases per 1,000 admissions for all causes).

Hospital and Date.	No Rice.					Cured Rice.					Fresh Rice.					Uncured Staple Rice.																	
	Europeans.*					' Bengalis. '					Tamils.					Malays.					Chinese.												
	Admissions.			Proportion of Beri-beri per 1,000.	(c)	Admissions.			Proportion of Beri-beri per 1,000.	(c)	Admissions.			Proportion of Beri-beri per 1,000.	(c)	Admissions.			Proportion of Beri-beri per 1,000.	(c)	Admissions.			Proportion of Beri-beri per 1,000.	(c)								
	All Diseases.	Beri-beri.	(b)			(a)	(b)	(a)			(b)	(a)	(b)			(a)	(b)	(a)			(b)	(a)	(b)			(a)	(b)	(a)	(b)	(a)	(b)	(a)	(b)
(a)	(b)	(c)	(a)	(b)	(c)	(a)	(b)	(c)	(a)	(b)	(c)	(a)	(b)	(c)	(a)	(b)	(c)	(a)	(b)	(c)	(a)	(b)	(c)	(a)	(b)	(c)							
Kuala Lumpur Pauper Hospital, 1891-1893	—	—	—	—	—	612	5	8	—	—	—	—	—	—	—	—	—	9,191	2,596	282	—	—	—	—	—	—							
Malacca Pauper Hospital, 1897	—	—	—	—	—	138	4	29	—	—	—	—	—	—	—	—	—	1,838	637	346	—	—	—	—	—								
State of Perak (all hospitals), 1899	—	—	—	—	—	5,019	6	1	—	—	—	—	—	—	—	—	—	14,166	2,811	198	—	—	—	—	—								
State of Selangor (ditto) 1899	—	—	—	—	—	2,332	16	6	—	—	—	—	—	—	—	—	—	9,070	1,720	189	—	—	—	—	—								
Negri Sembilan, 1891 to 1900 (ten years)	—	—	—	377	nil	3,368	16	4	—	—	—	—	—	—	—	—	—	7,884	3,428	435	—	—	—	—	—								
<i>Straits Settlements, 1899-1901.</i>																																	
Singapore General Hospital ..	1,530	nil	nil	1,361	10	1,485	62	41	—	—	—	—	—	—	—	—	—	772	44	57	—	—	—	—	—								
Singapore Pauper Hospital ..	387	7†	18	189	8	1,290	32	25	—	—	—	—	—	—	—	—	—	352	29	82	—	—	—	—	—								
Penang, General Hospital ..	591	5†	9	1,249	nil	1,109	7	6	—	—	—	—	—	—	—	—	—	1,072	10	9	—	—	—	—	—								
Penang, Lock Hospital ..	—	—	—	—	—	34	9	264	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—								
Penang, Pauper Hospital ..	148	2	13	129	2	3,012	23	7	—	—	—	—	—	—	—	—	—	115	2	17	—	—	—	—	—								
Penang, Balik Pulau Hospital ..	—	—	—	1	nil	296	2	6	—	—	—	—	—	—	—	—	—	44	nil	nil	—	—	—	—	—								
<i>Province Wellesley District Hospitals, 1899-1901.</i>																																	
Butterworth Hospital ..	—	—	—	—	—	1,289	nil	nil	—	—	—	—	—	—	—	—	—	200	nil	nil	—	—	—	—	—								
Bukit Mertajam Hospital ..	—	—	—	22	1	244	1	4	—	—	—	—	—	—	—	—	—	67	nil	nil	—	—	—	—	—								

Caledonia Estate, 1892-1902 ..	nil	—	—	—	nil	51	—	—	—	21,487	1	0'009	nil	72	—	—	23	nil	4	nil	12
Sungei Bakap Hosp., 1899-1901	—	—	—	—	—	—	—	—	—	1,373	nil	—	—	—	—	—	323	—	—	—	—
<i>Malacca, 1899-1901.</i>																					
General Hospital ..	3	nil	—	—	92	23	nil	nil	42	—	nil	nil	490	18	36	204	—	—	—	—	
Pauper Hospital ..	—	—	—	—	23	12	nil	nil	1,006	—	11	10	329	6	18	5,356	768	—	—	—	
Jasin District Hospital ..	—	—	—	—	—	—	—	—	155	—	nil	nil	51	—	—	2,429	280	—	—	—	
Alor Gajah District Hospital..	—	—	—	—	—	—	—	—	145	—	nil	nil	192	—	—	1,364	110	—	—	—	
<i>Pekang Hospitals, 1899-1901.</i>																					
Kuala Lipis General Hospital	7	nil	—	—	457	142	nil	7	335	—	4	11	293	4	13	831	161	—	—	—	
Raub District Hospital ..	—	—	—	—	1	1	1	7	285	—	2	7	176	8	45	457	132	—	—	—	
Bentong District Hospital ..	—	—	—	—	—	—	—	—	43	—	1	23	3	—	—	796	339	—	—	—	
Pekan District Hospital ..	—	—	—	—	133	—	1	7	44	—	8	181	145	2	13	223	126	—	—	—	
TOTAL ADMISSIONS ..	2,666	—	—	—	4,237	—	—	—	45,133	—	210	—	4,748	—	—	83,719	—	—	—	—	
TOTAL CASES BERI-BERI ..	—	148	—	—	—	23	—	—	—	—	—	—	—	144	—	—	16,325	—	—	—	
AVERAGE INCIDENCE OF BERI-BERI	—	5	—	—	—	—	—	5	—	—	—	4	—	—	30	—	—	—	—	196	

* Includes *Eurians* (mixed rice-eaters).

Two were 'Government subordinates'—probably Eurasians, one a European, the rest Eurasians.

§ This total includes, therefore, only two Europeans.

† One only of these was a European.

COMPOSITION IN REGARD TO NATIONALITY:

[illegible]

196 THE CAUSE AND PREVENTION OF BERI-BERI

same as their native resident neighbours, who were 'immune' from the disease, without, however, gaining the effect for which he had hoped. Vorderman mentions how young Javanese recruits are especially liable to beri-beri, incurring it immediately they leave their village homes and enter barracks, where, however, the rations given them are 'the same' as their own wonted diet.¹ A more instructive instance still is that which he gives of the Pradjoerits, or native militia, at Tijandjoer.² These men, employed as scouts, field-police, etc., outside the regular army, and furnished with military rations of Government white rice, came into barracks only while on duty. They lived and slept in the native villages. Here, it might have been expected they would remain free from beri-beri, as all the other villagers were. But the result was otherwise. The Pradjoerits got beri-beri, although the villagers did not.

Now, the only difference between these Pradjoerits affected by beri-beri and their native neighbours who were 'immune' lay in the rice they ate. Rupert's plan of making the conditions of his men purposely identical at all points with those of the villagers with whom they dwelt, again, failed in but one, and that the same, particular. It was not appreciated that there could be any distinction between the two sorts of rice. Yet those who ate stale white rice (the Government ration) got beri-beri; the native residents eating fresh rice did not. Between them there was no other difference.

Fiebig, in an exhaustive paper,³ sets out to show that the view commonly held and affirmed by Erni, Aquila, Van Lokhorst, Weintraub, Gelpke, Van Hoogeveen, Rupert, and Hirsch following these, that the indigenous natives of the Malay Archipelago were exempt or immune from beri-beri, so long as they lived free and after their own manner, is incorrect.

He brings his own observations, the reports of numerous medical officers, and abundant official records forward to prove that beri-beri does occur among such 'free' natives—that it is, in fact, endemic and liable at times to be epidemic in almost every part of Malaya.

It must be admitted that Fiebig clearly proves by the evidence he submits that there are few parts of the Dutch possessions or of islands beyond them in which cases of a disease apparently beri-

¹ 'Onderzoek,' p. 129.

² *Ibid.*, p. 61.

³ 'Beri-beri onder de dessabevolking in Nederlandsch-indie,' *Gen. Tijdschr. v. Noed.-Ind.*, Deel xxx., p. 432, by M. Fiebig.

beri had not been known to occur among the natives. The reports he relies upon are, however, in many of his instances, not those of medical but of other administrative officers, and it is at least highly probable that often the symptoms which they would attribute to beri-beri would be those of another disease. Almost any kind of dropsy is in the East dubbed beri-beri by the layman. 'Acute anæmic dropsy'—acute and massive anasarca, without any attendant paralysis, and without renal hepatic or cardiac mischief sufficient to account for it, and which moreover usually speedily and permanently disappears—is a disease which certainly is common enough in the Malay Peninsula, and may very well occur throughout the Archipelago. It has been the cause of much confusion elsewhere, and it is at least likely that it may have been mistaken for beri-beri in many of the instances which Fiebig records. But granting Fiebig's point to have been proved, supposing it to be a fact that cases, even epidemics, of beri-beri do occur from time to time among the free natives of all Malayan countries, who *usually* subsist on *usually* innocuous freshly-husked rice, such a result neither militates against the truth of our theory, nor is it necessarily opposed to the records of the observers who have asserted the *general* complete immunity of the Malay while living free and after the manner of his race.

For, of the theory itself it is naturally a corollary (and will be found among the deductions already enunciated), that even whole (unhusked) padi should be liable at times to contain such a quantity of poison (being heavily infested with the supposed fungus) that the rice made from it would entail beri-beri, even when eaten immediately after husking, and so nominally quite 'fresh'—though in no case was it *proved* to be fresh.

Moreover, it can be shown that many and often seemingly trivial factors may determine the production of the disease, so that cases of actual beri-beri among persons supposedly continuing in a normal course of life may after all have been due to unnoticed deviations from the usual conditions. Thus it may often happen among natives that drought restricting their crops, or pestilence their herds, storms damaging their grain, or poverty from any cause diminishing their means to buy the usual adjuncts to their food, the rice they eat is taken relatively in excess, not sufficiently supplemented by other food, or the stale rice of the shops is bought instead of the usual fresh grain.

Such conditions must happen, and will sufficiently account for

the occurrence of scattered epidemics of beri-beri among what are normally only fresh-rice eating communities.

They will not, however, weaken the statements of those observers who have dwelt upon the surprising anomaly of the disease attacking only new-comers into villages all whose native residents remain exempt. Nor will they invalidate the truth of the general statement that the Malay everywhere, *so long as he eats only fresh rice*, remains free from beri-beri.

Tradition, it would seem, has taught the 'native' races everywhere more wisdom than the civilized possess in regard at least to the use of rice.

Not only in Malaya proper, but in Madagascar, in Burmah, in Siam, in China, the inland natives always eat rice fresh.

Evidence from Madagascar.—In Madagascar Pétit¹ records that beri-beri attacked the Senegalese troops, fed on Government (white rice) rations, severely at many stations, but 'not a single case was ever observed among the native inhabitants of the villages adjoining these stations, and this, I believe, is due to the fact that they keep their rice stored in the husk, hulling it day by day as required for use.'

His observations firmly convinced him that beri-beri was due to stale rice.

The Malagasy is, however, susceptible (as are all races) to beri-beri.

Schuttelaere² gives account of two epidemics at Diego-Suarez, a port on the north coast of Madagascar, among both native troops and civilians in 1900.

The first was 'a small epidemic of beri-beri which appeared among the Betsileo guides—black Malagasies—of the marine artillery; it was a question of a few mild cases only. The diet of these natives contained no fat. I had 30 grammes a day allowed them, and no more cases happened.'

'Later, in October, 1900, an epidemic of different severity showed itself. . . . This, which lasted about six weeks, furnished 180 cases, and caused 37 deaths.' The disease first appeared in a company of Senegalese, who had arrived but three months previously. It was ushered in, as commonly happens, by several unexpected and sudden deaths in persons only slightly or vaguely ill. Then other cases, typical pictures of paresis and œdema, dry, wet, and mixed beri-beri appeared. There was no doubt

¹ *Ann. d'Hyg. et de Méd. Col.*, 1903, vol. vi., No. 1, p. 98.

² *Arch. de Méd. et de Pharm. Mil.*, t. xxxvi., No. 12, December, 1901, p. 470.

about the diagnosis, which was concurred in by several officers who had had experience of beri-beri in the East.

The men attacked were healthy, young, well fed, well lodged, lightly worked. Their ration included 400 grammes fresh meat (buffalo meat is as cheap as rice in Madagascar), and 30 grammes of fat, so there could be no question of nitrogen or fat-starvation. At Ankerika, the station at which these troops were, they were the only troops attacked. Among two companies of Creoles, from Reunion, there were no cases. Two other companies of Senegalese only recently arrived (three weeks) also were not attacked.

But, later, a few cases appeared among other companies: some among the more lately arrived Senegalese; some among the Senegal guides at Sakarary, a post in the interior 450 metres high; others in Chinese labourers; among Malagasies in prison especially; and some rare cases among the Reunion Creoles—the darker ones—who ate chiefly rice. Schuttelaere had the rice of all the troops using it reduced by one-half from November 5, and the Senegalese troops removed to another station. 'From this moment the gravity of the epidemy declined; but the number of cases remained high among the Senegalese who had remained at Ankerika. It should be said that the general *visites de santé* had revealed many mild cases in men who complained of nothing; a minute examination showed slight œdema of the legs and acceleration of heart-beat, sure indices of the malady beginning. . . .' 'And, moreover, beri-beri continued to prevail among all the rice-eaters whose ration, in respect to this article, had been reduced to half.' 'The rice issued to the troops and civilians came from Indo-China; it had already arrived some time: it was a bit old.'

'The native Malagasies do not eat the Indo-Chinese rice except when they cannot help doing so. They distrust it, preferring fresh rice grown in their own country.'

The more radical measure was then adopted of 'the complete suppression of the rice in the native ration, and the substitution for it of bread, and *pain de guerre*; later by *paddy* when one could.'

'On November 21 this measure had come fully into force, and the epidemy was extinguished at once; no new case of beri-beri appeared, except among the Chinese coolies, who, *bêtement routiniers et traditionnalistes*, would not renounce the rice derived from their own country: they struck work if one did not give

them this; beri-beri continued to make victims among them.' 'At the end of a month the Ankerika station was reoccupied by the Senegalese, henceforth deprived of rice; beri-beri did not appear again.'

Commenting on the whole account, Schuttelaere says: 'It appears, then, that the series of facts just represented has all the force of a decisive experiment, without, however, fully satisfying scientific curiosity, since more than one point remains obscure.'

He refers here to the apparent immunity of the half battalion of Senegalese who arrived in the country only a few days before the disease broke out, while their fellows were being severely attacked. But this was clearly due to the process of intoxication not having in the former case endured long enough.

Instances to the same purport, illustrating the difference in effect of eating stale as compared with fresh rice, might be largely multiplied, and more of them will be found in other sections, the particular arguments in which they better enforce.

Evidence from Java Gaols.—But there has now to be presented the evidence of experiments made with another 'sort' of rice—namely, 'red rice,' the remarkable and important results obtained with which are the achievement of Vorderman.

Stimulated by Eijkman's declaration, that polyneuritis in fowls was produced by white rice, and cured by red, and reflecting on his own experience of beri-beri, which only appeared in gaols in Java where white rice was eaten, Vorderman set afoot an elaborate investigation into the relation of these two sorts of rice to the disease in all the prisons of Java and Madoera.

The observations were carried out in 1895, 1896, in 101 prisons, of which all the inmates were fed for the same length of time—eighteen months—and simultaneously, on one or the other of the two sorts of rice, *red*—with as much pericarp as possible preserved—and *white*, with as much of it as possible stripped off. At some prisons a mixture of the two sorts was supplied. The rice was in every case, where this was possible, of the sort grown locally, but supplied through contractors.

This was the result:

There passed through the 101 gaols during the period of experiment 281,878 persons.

There were 96,530 prisoners interned in gaols where only *red rice* was used. Among them occurred *nine cases of beri-beri*, or about 1 in 10,000.

There were 150,266 incarcerated in gaols where only **white rice** was used. The result was 4,201 cases of beri-beri—a rate of nearly 28 per 1,000.

Those, 35,082 in number, kept at prisons dieted on the mixed ration showed a result intermediate between these two—*i.e.*, 85 cases—2.4 per 1,000.

Of the 101 gaols, white rice was used at 63, and beri-beri appeared at 34 of these, or 54 per cent.

Red rice was used at 27, and there was beri-beri at 1 only of these. Mixed rices were given at the remaining 11 gaols, and at 6 of them (46 per cent.) there was beri-beri.

Elaborate and minute inquiry was made into all other sanitary and hygienic conditions affecting the gaols, and the results are set forth at length by Vorderman. It was impossible to trace whether in locality, or height above sea-level, in ventilation, or overcrowding, in structure or condition of buildings, or in sickness-rates from other causes, any feature which indicated any influence whatever, due to any of these factors, upon the prevalence or varying case-incidence of the disease.

It should be added that there was no distinction as to either races or classes of prisoners confined at the different gaols, and fed on different sorts of rice. All except three of the prisons were 'district prisons' of a common type.

It was not only at the prisons that this astonishing protective effect of the use of red rice against beri-beri was noted. Vorderman records many remarkable instances of the same difference entailing the same effect elsewhere.

1. Thus, at **Kediri** there was a lock hospital, which was usually overcrowded. Here in June, 1896, beri-beri broke out. The food supplied to the inmates had been for a long time of local unscoured ('red') rice; but this was changed by the contractor to white Saigon rice, 'which, when cooked, has a better flavour.'¹

A short time after this change beri-beri suddenly appeared among the prostitutes. In a few days there were 22 cases in a daily average strength of 113 persons. Vorderman had the Saigon white rice substituted by local unstripped rice, half white and half red, such as was in use by the local natives. Thereupon the disease declined.

2. At the District Prison, **Soerabaya**,² while white rice was used there were, during the last five months of 1895, 29 cases of beri-

¹ 'Onderzoek,' p. 108.

² *Ibid.*, p. 123.

beri among 288 prisoners. During the corresponding time in 1896 red rice was used, and there were among 407 prisoners only 3 cases. A reduction of the case-incidence from 66 per cent. of daily strength to 4 per 1,000.

3. At the 'Pegiriang gesticht' Lock Hospital¹ there were in 1895 16 cases among 161 inmates on white rice. The following year, on red rice, only 2 cases appeared among 133 inmates observed for a corresponding term.

4. **Karanganjar** was a prison which had always used red rice, and where there had never been a case of beri-beri.

Two convicts were sent from this gaol to do work at a neighbouring military hospital, that of Gombong. They lived and slept there. The usual gaol-ration was given them, with the difference that for the red rice used at the prison white, stripped rice, being the sort in use at the hospital, was substituted.

Both convicts were invalided at the hospital as unfit for work, and on returning to the prison were found to be suffering from beri-beri.

5. **Magelang** was another gaol at which red rice had always been in use, and at which there had never been beri-beri.

A detachment of prisoners from this gaol worked at the large military hospital of Magelang—lived and slept there. Their diet again was the customary prison ration, with white rice, such as was used at the hospital, substituted for red.

At the end of 1895 one of these prisoners fell sick with beri-beri, was sent back to the prison, there fed on red rice again, and recovered.

Later, in 1896, seven more of the detachment of convicts got beri-beri at the hospital. One, severely attacked, was kept at the hospital with a view to the better treatment which might be afforded him, and there, dieted on white rice, he died. The other six sent back to the prison, and fed again on red rice, all rapidly recovered.

Now in regard to the detachment of convicts at the military hospital, Pekelharing and Winkler had remarked, says Vorderman, that prior to 1895 not a single case of beri-beri had appeared among them, although they were in constant association with beri-beri patients suffering from relapses or recurrences, or established cases sent in from other places. They also, incidentally, stated that the prisoners in the detachment at the hospital at that time were fed on prison-rations and with *red* rice.

¹ 'Onderzoek,' p. 128.

The point which these authors wished to make was that beri-beri in its chronic or recurrent form was not infectious, but in acute form probably was so. Although from 1890 to 1895 many chronic cases had been in the hospital with which these prisoners had had contact, all that time no case of the disease had appeared among the latter. But in 1895 three primary cases arose among soldiers of the garrison, which were treated in the hospital, and it was after this that the prisoners first became attacked. Vorderman himself, however, discounts such significance as this coincidence might have been held to possess by the observation that the primary cases which arose among the prisoners themselves sent back to the prison certainly entailed no ill-results among the rest of their fellows in that institution.

The point of present value in the incident is that it demonstrates the dependence of beri-beri upon the sort of rice eaten under conditions almost equal to those of designed experimental control. For the convicts who acquired beri-beri did so only after their customary red rice had been changed for white at a place where for many years previously so long as their rice was red they had been proof against the disorder, and at a time when their fellows (at the prison), fed on an identical diet save that their rice was not white but red, continued to be exempt from beri-beri.

That the convicts who got beri-beri at this hospital did so because they were fed on white, stripped, instead of unstripped red rice is obvious. Why the patients at the hospital itself and all the rest of the very numerous garrison, who all shared the same toxic rice, did not get the disease also, may be better explained in the next section.

These and many other facts related by Vorderman establish the conclusion that the use of 'red' rice prevents beri-beri.

The importance of his discovery was fully appreciated by Vorderman, who urged, and in many cases secured, the substitution of red rice for white wherever possible in the public institutions of Java. But he offers no explanation of the rôle played by the pericarp—the presence of which on red rice seems to be the only difference he admits between it and the white—in the prevention of beri-beri.

That it could not be merely antidotal to a poison in white rice, or to symptoms produced by eating white rice, the exceptional occurrence of beri-beri at Bangkalan, one of the gaols fed only on red rice, seems to show. That beri-beri could, in any case, not depend solely upon white rice, its non-occurrence in

nearly half of the gaols fed on seemingly the same white rice, as well as its exceeding variability, seemed to him equally a proof. 'In the production of beri-beri there must thus,' he says, 'be several factors at work, among which I would, because of the infectiousness of the disease, place the operation of micro-organisms.'

It is notable that, with all his experience, and holding these views, the only fact put forward by Vorderman as evidence of the infectiousness of beri-beri is the occurrence of the disease at certain prisons—4 out of 101—where it happened to break out soon after the admission to the gaol of one or more primary cases of the disorder incurred without. It weighs nothing against such reasoning that there have been thousands, indeed hundreds of thousands, of such instances of the admission of primary cases among unaffected people—the remaining thirty Java gaols on white rice at which it prevailed among them—without the disease so spreading to others.

When the protection afforded by the use of red rice is viewed in the light of the other facts already brought forward in this work—the equal protection afforded by the use of fresh or cured rice—a simple explanation of the result becomes obvious.

None of these sorts of rice, strictly speaking, does protect against beri-beri, in the sense of acting antidotally. The fact is simply that none of them conveys or causes beri-beri, as stale uncured white rice does. Red rice comes into the category because it is, so long as the pericarp remains on it, protected against the change which stripping off the envelopes usually entails in all except cured rice. It is, in fact, to all intents and purposes still unstripped padi—*i.e.*, it is fresh rice. Vorderman's very exceptions make for this view.

Absolutely pure red rice—*i.e.*, samples in which every grain retained all its pericarp—was not used in any of the gaols. Rice was termed 'red' if it contained not more than 25 per cent. of decorticated grain—*i.e.*, stale white rice—mixed with it.¹ Now, it is certainly possible that at times white rice should become so intensely toxic that a proportion as small even as 10 per cent. in the whole bulk should produce bad effects. Moreover, by our theory, the poison or its agent may, under rare favourable conditions, be present to an unusual extent even in *fresh* rice. It is no matter for surprise, therefore, that one among all the prisons

¹ 'Onderzoek,' p. 58.

fed on 'red rice' in Vorderman's experiments should show beri-beri.

The reversed conditions apply to the cases where prisons fed on white rice did not show beri-beri. It is stated in Vorderman's tables of results that the 'white rice,' so called, might contain as much as 25 per cent. of red.

Now, to anticipate the argument of the next section, there is evidence to show that the *dilution* of stale rice which would otherwise prove toxic, whether by fresh rice or any other aliment, to an extent of 25 per cent. (or less) will often prevent beri-beri following. Therefore, it might be expected, on this ground alone, that some of the prisons in which white rice so diluted was used should show no beri-beri.

Vorderman gives the case-incidence of beri-beri in respect to the total number of prisoners passing through the gaols concerned for the whole period, excluding political prisoners, who were in for very short intervals only. But, as Van Dieren pointed out, the more correct way of representing the facts would be to give a case-incidence according to the daily average strength of the prisons, and expressed in this way the result is even more startling. The following table is thus compiled by V. Dieren¹ from Vorderman's figures :

	37 Prisons on Half-peeled Rice.	13 Prisons on Mixture.	51 Prisons on Wholly-peeled Rice.
Average strength - - -	6,295	2,040	9,496
Total cases beri-beri - - -	9	80	4201
Case-incidence beri-beri - - -	0.14%	4.1%	44%
Total deaths beri-beri - - -	4	22	752

Van Dieren severely handles the statistical methods of Vorderman, yet the main results obtained by the latter stand unimpaired, and seem, indeed, unimpeachable. But his conclusion that 'the cause of its appearance [beri-beri] is thus not to be ascribed exclusively to the nature of the staple diet,' Van Dieren points out, should more strictly be expressed in a mode implying that the disease does not depend exclusively upon the pericarp of the rice.

Van Dieren further shows ('Kantteekeningen,' pp. 37, 38) that the conclusion which Vorderman reached, that the *source* of the white (wholly stripped or peeled) rice used in prisons where

¹ *Loc. cit.*, p. 42.

206 THE CAUSE AND PREVENTION OF BERI-BERI

beri-beri resulted from its use was immaterial, or without marked influence, is incorrect. When the case-incidence is calculated according to daily strength of the various gaols, instead of in proportion to the total prisoners incarcerated as by Vorderman, it appears that there was a very marked difference in the effects of local (home-grown) and imported (Siam, Rangoon, or Saigon) white rice. The results in tabular form were as follows :

	27 Prisons on Home-grown Wholly-peeled Rice.	19 Prisons on Imported Wholly-peeled Rice.
Total strength - - - -	4,834	3,923
Deaths from beri-beri - - -	235	517
Deaths from other causes - -	203	403
Mortality from beri-beri - - -	4.8	13.2
Mortality from other diseases -	4.2	10.2

A third factor, the importance of which has been dwelt upon in preceding sections, which might explain many apparent discrepancies in Vorderman's results, is the prolonged latent period usual in beri-beri. Vorderman, it is true, records observations to show that this is generally lengthy (he assigns an average of 106 days), and he shows (pp. 101, 102) how the incidence of the disease when it did occur was directly proportionate to the length of sentence served by the subjects. But no records are given of the different numbers of individuals undergoing sentences of different lengths in the prisons under observation. In their absence it may well be assumed that, when beri-beri failed to appear in certain prisons fed on white rice, this was because there were no prisoners (or too few of them) serving terms of sufficient length to cover the necessary period of latency.

The proof thus afforded that it is on the sort of rice eaten that beri-beri depends enables many epidemics to be understood, the peculiarities in the distribution of which had previously been unaccountable. These in their turn afford additional evidence of the same tenour.

It has been said that in China, Siam, and Burmah the natives eat fresh rice. Their almost entire exemption from beri-beri has long been known—has, indeed, endured through all historical times—so that there is no word for beri-beri in either vernacular. But at the present day cheap ready-made rice prepared in the mills—the stale uncured sort—is, as one of the mistaken blessings of civilization, steadily displacing fresh, and coinci-

dently beri-beri is in all such places yearly becoming more prevalent, especially in large towns and along the coastal areas, which are readily supplied from ports, and where, moreover, the inhabitants, usually fisher-folk, do not grow padi and make their own rice from it.

Evidence from Siam.—Beri-beri has been believed to be especially rare in Siam, where the people, mostly Buddhists, eat indeed little meat, but plenty of vegetables, with fresh rice. But there was an outbreak in 1900 at Bangkok.

Of this epidemic I am fortunate in being able to publish the following notes, with which Dr. Nightingale, lately the principal medical officer there, has most kindly supplied me from memory :

‘ The first case of beri-beri (of the epidemic) I saw in Bangkok was early in November, 1900. It was that of a police constable in charge of the telephone at the Local Government Board’s office. He was a very robust man, who lived day and night in the telephone-room on the second floor of the building. *He was in the habit of buying his food from an itinerant Siamese pedlar, who supplied many others in the building in the same way. After office hours he (the pedlar) sold the curry and rice in the streets to coolies and others.*

‘ When the constable came to me (as police surgeon) in the same building, he complained of shortness of breath and slight swelling of the legs. He did not know what was the matter with him, nor did his friends. He said he knew it was not malarial fever, and was *firmly* convinced that the telephone had bewitched him. About three weeks later he was admitted to the police hospital, where he died very suddenly from heart failure.

‘ About the end of November, 1900, I received reports that a mysterious disease was spreading along the sea border to Siam from Cambodia, causing numerous deaths. At first the Siamese authorities in Bangkok thought it was the bubonic plague, but on making inquiries I was convinced it was beri-beri.

‘ During December a considerable number of police were sent into the police hospital suffering from the disease. They came not only from Bangkok itself (which has an estimated population of half a million), but from many of the outlying stations up to twenty miles away. These men in the various stations fed themselves, and were not supplied with rice by one or more contractors.

‘ In December, 1900, and January, 1901, a large number of

208 THE CAUSE AND PREVENTION OF BERI-BERI

men in the navy, army, and gaols were attacked. These were fed by three sets of contractors, who probably bought considerable quantities of rice from the different mills.

'At the end of January, 1901, I made an official report on the beri-beri in the army, carefully examined their rations, sleeping quarters, etc., but could make out no cause for the epidemic. I advised that every man who was attacked should be dismissed from the force, and returned to their up-country homes as soon as they were fit to travel.

'The mortality was considerable, though no reliable statistics were forthcoming. Many others became hopeless paralytics of the well-known type.

'After February, 1901, the epidemic began to disappear as mysteriously as it had come. By June it was quite the exception to note any cases in the streets. The admissions to hospital had almost entirely ceased by that time.

'The only reliable figures dealing with the epidemic are to be found in the records of the police hospital, which I made myself. In this epidemic the Siamese were attacked in much larger numbers than the Chinese. For the most part they lived off a *better kind of milled rice* than the Chinese. The Chinese coolies in the rice-mills, saw-mills, ships, and general labour of the town did not suffer to the same extent, as far as is known, as the Siamese in the navy, army, police, and gaols. Though no definite figures were procurable, there can be no doubt that the disease attacked many of the country Siamese along the south-eastern coast—that is, people who would grow and hand-mill their own rice.

'There are about a dozen or fifteen large steam rice-mills in Bangkok, which work all the year round. From July to November is their busiest time, as fresh supplies of rice then come in, when the various canals are full of water from the heavy rains.

'*There are about 2,000 natives of India in Bangkok. They always feed off rice imported from India—rice that must have been milled at least one month previously.¹ Beri-beri among these Indians was very rare.*

'*Husking padi by hand mill-stones in Bangkok is yearly getting rarer.* This method, which still exists in many of the large establishments of the nobles for their family use, produces a fine type of unbroken white rice. I know definitely that some cases of beri-beri existed among the servants of these nobles, though I could not say what rice they had eaten as a rule.

¹ *Stale* therefore, but being prepared in the Indian manner not *uncured*.

Unless careful experiments were performed I should think that it was very difficult to differentiate between "stale uncured" and "fresh" rice, and am not aware that the method you mention of preparing "cured rice" exists in Siam.

'I cannot suggest any cause for the Siamese epidemic, and know that no steps were taken to combat it, and yet it passed completely away within five or six months, and there has been no recurrence of it during the last three and a half years. Of course, I believe that, under certain unknown conditions, it is very infectious.

'During my ten years' (1891-1900) tropical experience in Johore, Malay Peninsula, and Siam I saw a very great deal of the disease, and wrote my M.D. (Edinburgh) thesis on it in 1898. I must honestly confess that I am not a believer in the rice theory, *per se*, as a cause of beri-beri, though I can offer no definite theory in its place.'¹

It will be seen that some of the facts noted by Nightingale offer singular confirmation of the views as to the mode of production of beri-beri here advocated, and even those features which seem at first to militate against the 'rice theory' are just such as, happening elsewhere, have been found to be easily resolved when full details were forthcoming.

This incidence of the disease first or commonly upon those fed by contractors, whether as pedlars or on a larger scale (army, navy, gaols), is in correspondence with what happens in the Malay Native States and Straits Settlements. The peculiar liability of police at out-stations to suffer—exactly paralleled by the conditions again of the same class in the Native States—no doubt depended on the fact that, because isolated at such stations, and occupied in duty, they lived, as do the Malay police, not on home-prepared *fresh rice*, but on stale uncured rice bought at shops and then stored in quantity.

The greater relative proclivity of Siamese *eating a better kind of milled rice* than the Chinese exemplifies possibly the influence which the complete 'scouring,' 'polishing,' of the commercially higher-class product has in degrading its nutritive value. Also, probably, the Siamese, better off as a class, could afford to buy and eat more rice absolutely than did the Chinese coolies in the shops and mills. The latter, again, able, as free coolies, to supplement their rice by ordinary adjuncts, especially fat pork, would naturally, for that reason alone, suffer less than the Siamese,

¹ The italics in the above are the present writer's.

whether in navy, army, police, and gaols, fed on contract supplies and fixed rations. Moreover, it is to be remembered that Siamese as Buddhists, forbidden to take animal life, are, as a result, always less well supplied with meat and flesh than the Chinese, who are under no such restrictions.

The entire exemption of Indians using their own sort of rice specially imported from India is remarkable, and accords with the experience of the Federated Malay States related elsewhere. But even did it stand alone, this feature of the epidemy would demand attention, and should surely be held significant of some relation between the sort of rice eaten and the causation of beri-beri.

These facts from Bangkok are evidence all the more valuable in that they are recorded by an observer who frankly proclaims his disbelief in the theory which alone can explain them.

Evidence from India.—In India, beri-beri comparatively rarely happens, and the districts where it does occur are those in which 'curing' the rice, which is a method chiefly of Bengal, is not practised. In the Indian Army it has always been among the troops fed on rice—*i.e.*, those of the Madras command—that the disease has chiefly appeared, as it is solely among these troops that it continues to occur at the present day.

In the gaols of India—at least, in those of Bengal and Madras in marked contrast with those of Burmah—beri-beri seldom or never appears.¹ In these gaols the greatest care is exercised, both in the selection and preparation of the rice used in the rations. In the first place, only the winter crop is used; that of the autumn is definitely forbidden. No padi which is less than three months old is taken, and the rice is made from the padi in the gaols by the curing process. In Burmah the rice given to the prisoners is the rice of the country, uncured.

¹ There was an outbreak of beri-beri at the large Rajahmundry Prison, Madras, in 1889, an account of which was given by Bidie (*British Medical Journal*, July 20, 1889, p. 115). The cases occurred among Burmese prisoners, of whom thirteen died. At that time the diets were supplied (Dr. Galdanha, the present Superintendent, informs me) by contractors, and the rice used was the ordinary bazaar commodity, which, as it was prepared in the district, was not cured—*i.e.*, it was of the white stale sort. In 1898 there was an outbreak of disease, as to the nature of which there was much discussion, but which was affirmed by Fearnside to be beri-beri. Although there were many and some fatal, cases, the pictures of pronounced atrophic paralysis common in other epidemics of beri-beri do not seem to have been produced here. Since 1889 this gaol has been fed departmentally, and the rice given is freshly husked, but not cured.

In Calcutta, if beri-beri has occurred in the past in great epidemics, it is not now a disease of importance, but it continues to occur sporadically. The majority of the inhabitants, perhaps, do not eat rice, but there are at least many thousands that do. But among these the disease is limited to quite a small class of aliens.

Major Buchanan, to whom the writer is indebted for this information, says: 'In Bengal, where 70,000,000 eat rice, beri-beri is unknown, and in Calcutta you will only see it among Chinese carpenters.' Now, these people, who are as conservative in some of their habits as are the Indians themselves, dislike cured Bengal rice, and import supplies of the kind they prefer—uncured Siamese or Rangoon rice from the outside, principally from the Straits Settlements.

In Rangoon, Barry describes beri-beri as affecting almost entirely Indian coolies from the South, the very class who are referred to in this article as Tamils, who, in this region where beri-beri is rife, have been shown to enjoy almost entire immunity from the disorder. Barry's observations led him to the conclusion that the probable cause of their getting beri-beri in Rangoon was excessive addiction to drinking *rice-kanji*, the liquor in which rice has been boiled. Rost, further investigating the same subject, says he is 'quite sure' that the disease develops from drinking rice-water liquor, or from feeding on diseased rice. He believes that Tamil coolies become affected in Rangoon because they drink largely the fermented liquor made from rice. Cases occurring among European troops and Eurasians in Rangoon he ascribes to the same habit. It is plain that this view of the causation of beri-beri in the limited class of persons who acquire it in Rangoon accords well enough with the theory developed in this book. But it is not necessary for such a result that the immediate vehicle of the poison should be rice-liquor, either plain or fermented.

The Tamils who get beri-beri in Rangoon are natives who leave India in large numbers annually to assist in the rice-harvest in Burmah.

This immigration takes place in September. In an account of the circumstances of these coolies in the 'Notes and Statistics of the Hospitals and Dispensaries of Burmah for 1899,' the writer—Leslie—states that there is no privation among them, and that it is generally from nine months to one and a half years after arrival in the country that they succumb, first to fever, and then to beri-beri.

212 THE CAUSE AND PREVENTION OF BERI-BERI

Now, these immigrants are independent. It does not appear that they are looked after by contractors or large employers, or in any way cared for or provided with special food and management by legislation, as are the Tamil immigrants into the Straits Settlements.

It follows that they subsist on their own means—that is to say, on the cheapest food which they can procure, which is naturally the rice of the country as sold in the shops—*i.e.*, uncured and stale. Their food is, in fact, precisely the sort upon which, when they are fed, even in the British Malayan Settlements, as happens in the gaols and asylums, they acquire beri-beri.

Upon what *essentially* the diverse effects of consuming these different rices may depend will be discussed later. In this section it has been sought only to prove, as it will perhaps be admitted has been done, by evidence too clear and too extensive to be denied, that, as predicated by the theory, *the incidence of beri-beri, everywhere throughout British Malaya, and wherever the circumstances have been investigated, varies with the sort of rice eaten.*

SECTION IV (*continued*)

THE CASE-INCIDENCE AND MORTALITY OF BERI-BERI VARY DIRECTLY WITH QUANTITY OF RICE EATEN

III

BERI-BERI PROPORTIONATE TO QUANTITY OF RICE EATEN

The Third Deduction—that Beri-beri should vary with 'quantity' of Rice Eaten.—The third among the conclusions drawn from the theory, and deducible from no other premises, was that *the incidence, and even the mortality, of beri-beri should vary directly with the quantity, absolute or relative, of rice eaten.*

Evidence in British Malaya.—Owing to the fact that the assumed poison in rice which produces disease has not yet been identified, precise evidence on this head is not obtainable. In the absence of such definite knowledge of the toxic principle, and consequently of the quantity which may have been taken by persons, or of the period during which it may have been in consumption, it is impossible to make even an approximate equation between the effect and the absolute dose producing it.

But if it were possible to place two different sections of a population otherwise all equally susceptible to beri-beri for sufficiently prolonged periods upon a diet composed chiefly of the same uncured rice, but in quantities which should, for whatever reason, always be unequal, either absolutely or relatively, or both, for the two classes, then the result would afford some measure of the direct effect which quantity of rice eaten has upon the incidence of the disease.

I. Evidence from Chinese Immigrants into British Malaya—Incidence of Disease varies amongst Different Classes.—Such a state of affairs is realized in British Malaya.

214 THE CAUSE AND PREVENTION OF BERI-BERI

In this region, as the writer's analysis of 140,503 unselected cases of sickness consecutively treated during recent years in Government hospitals proves, it is the Chinese who provide almost all cases of beri-beri.

Of 16,716 cases of that disorder tabulated, they furnished 976 in every 1,000, or all but $2\frac{1}{2}$ per cent.

If investigation be made of the circumstances of these Chinese, it will be found that they are, through the operation of economic causes, divided into sections, the habits of which as regards quantity of rice eaten are quite distinct.

These divisions are, in the first place, those of (1) the employers, and (2) the employed.

The Employers are least Affected.—In British Malaya there is little to distinguish these two classes. In physical proclivities, appetites, and aptitudes, in mental and moral tastes and tendencies, in matters of personal hygiene, in customs of cooking and feeding, of clothing and cleanliness, there is no difference between employer and employed. If any, it is in favour of the coolie, whose strenuous labour makes his body vigorous, and ablutions a pleasure, whereas the wealthier employer becomes gross and indolent, and goes unwashed. There is even little difference in education between the labourers and the headmen. Most of the latter have risen to their position, however, through force of greater industry or intelligence. A superior leisured or learned class of Chinese is not represented in the Native States. The only social distinction is that defined by the possession of more or less wealth.

Even this factor does not entail any degree of alteration in the manners and customs, or diminish the extent of contact of the two classes. In every phase of commerce and industry the association between both is of the closest. In mines, on agricultural estates, in shops and on ships, even in the private houses of the rich, employers and employed are always in close and intimate contact, sharing and equally exposed to all such circumstances as could be conceived capable of modifying the possible incidence of disease.

With one exception.

The position of employer, the possession of relatively greater wealth, brings it about that those who have it are able to indulge in a more liberal and varied class of diet.

The 'towkay,' or headman, thus eats plentifully of fresh meat, generally very fat pork, eggs, fresh fish, fruit, vegetables, and

sweetstuff daily, all of them articles which expense renders inaccessible to the coolie. The latter may obtain these luxuries upon feast-days, or other rare occasions, but for nine out of every ten days his diet consists of little more than rice flavoured by curry-stuff, a little salted fish, some peas, black beans, or other pulse.

However much rice the towkay may usually consume absolutely—and it may be more in some cases than is eaten on an average by the coolie—the quantity eaten by towkays as a class is thus *relatively* less in proportion to the other components of the diet than is consumed by the coolie. In accordance with the theory, therefore, it is to be expected that the incidence of beri-beri upon the former class who eat less rice should be smaller than it is upon the labouring classes who eat more. This will be shown to be the case.

In the Straits Settlements, although of the half million or more Chinese found there, the majority are but temporarily resident, yet there is absolutely a large number permanently settled, many being native-born. The number of these Straits-born Chinese in the Colony is over 50,000, of whom some 11,000 are male adults. Most of them are well-to-do, all of them better off than coolies. They are engaged principally in commerce, counting-houses, etc., and are to be reckoned as certainly belonging to that section of the population which has been defined as eating relatively less rice than the coolie, and therefore as proportionately less liable than the latter to get beri-beri.

Now, in the Colony and States better-class natives who seek admission to hospital are treated in the 'General' (paying) wards of the hospitals at each place, coolies in the pauper wards. The relative proportion of beri-berics among Chinese treated at such institutions will afford an index, therefore, of the extent to which each class is liable to the disorder.

Reference to the preceding table (given on p. 195) shows that at Singapore during the three years 1899-1901, of 16,045 Chinese treated at the *Pauper* Hospital, 113 in every 1,000 had beri-beri, but of 5,062 Chinese treated at the *General* Hospital during the same period only 51 per 1,000 had it. At Penang, at the same time, the proportion of beri-berics to all Chinese treated was 124 per 1,000 at the Pauper Hospital, and 44 per 1,000 at the General Hospital. At Malacca the rate was 143 per 1,000 paupers (out of 5,356 patients), while not a single one of 200 patients treated at the General Hospital had beri-beri.

The greater relative incidence of the disease on the poorer class seems sufficiently proved by these figures. Yet they indicate but partially the really surprising degree of freedom from beri-beri which is everywhere enjoyed by the towkay class as compared with their coolies, and which has impressed itself as remarkable upon all those who, like the writer, have had an intimate acquaintance with the conditions under which beri-beri prevails. Scores—nay, hundreds—of instances have come under my observation in which both at mines, and on estates, the coolies working under one and the same headman have, year after year, been afflicted, decimated, and even almost wholly extirpated, by beri-beri, yet the towkays living at the mine, occupying the same building even with the coolies, and sharing with them every imaginary or actual insanitary influence in operation about them, have remained permanently unaffected.

Among others, H. Wright has noted the immunity especially of the free Straits-born Chinese. He says: 'The Chinese are the greatest sufferers from beri-beri . . . the Straits-born Chinese, who represent the higher class of the Chinese communities, are the exception. They are not wholly exempt, but there certainly is some factor in their usual social and other habits that renders them more immune than other classes.' What that factor is has been indicated. But it is more interesting to note a second remark by the same observer, supporting the view that the 'immunity' of Straits-born Chinese depends upon their relatively smaller consumption of rice. 'In the Kuala Lumpur Gaol,' Wright says, 'what is almost unknown outside the gaol, the Straits-born Chinese are victims in as large a proportion as their fellows.' In the gaol, it appears, in a party kept under observation for some eleven months, there occurred, among 1,048 native-born Chinese, 70 cases of beri-beri; among 44 Straits-born Chinese 7 cases—proportions of 66 and 158 per 1,000 respectively. The numbers are, of course, too few to establish a generalization. But the observed fact supports the assertion that the usual exemption of the Straits-born is due to a relatively smaller consumption of rice, since, when this condition is reversed (as in gaols, where the diet is the same for all), he is attacked by beri-beri to a much greater degree than others of his race.

Durham,¹ who has noted and been impressed by the extraordinary exemption of the towkay, as compared with the coolie, has also observed that the only distinction between the two classes

¹ *Journal of Hygiene*, January, 1904.

is one of wealth and scale of diet. Unappreciative of the real factor—the different amount of rice consumed by the two classes—Durham attributes the result to a ‘sheltering’ effect produced against beri-beri in those who are better fed. He divides the rations eaten into ‘insufficient,’ ‘sufficient,’ and ‘supersufficient,’ but what this distinction may be supposed to import the writer, at least, fails to understand. Since Durham entirely rejects the view of production of beri-beri through food. But in that case his clear association of liability to beri-beri with different diets is a barren observation. Of what disease can it be affirmed that, although an infection, it *never* attacks those who are overfed, as his own observations show is the case in beri-beri? On the other hand, if a liberal diet ‘shelters’ from the disease, how explain the fact that the sickly, the starved, and the indigent are *less* liable to it than vigorous and robust and amply fed coolies?

Amongst the Coolies the Different Sections are Differently Affected.—Next, the coolie class itself, composing the great bulk of the population, and supplying at least 95 per cent. of all the cases of beri-beri, is again divided into sections by clear and actively operating distinctions, upon which important dietetic differences depend.

The bulk of the Chinese in British Malaya form a tidal element, which, continually depleted by deaths and emigrations to China, is as constantly supplied by immigration of fresh individuals. The numbers thus passing to and fro amount to hundreds of thousands yearly. Natural economic factors, chief among which has been the increased profits derived from the main product of industry—tin—have caused the flood of immigrants to swell yearly. The population, numbering 321,126 in 1891, was 474,282 in 1901—an annual addition of some 5 per cent.

Of those who enter the country for the first time, all but a small proportion arrive under contract to work for a term of from six months to two years, generally at a mine, or upon some plantation.

These men are termed ‘*sinkhehs*,’ literally ‘new-comers.’ They receive an advance of cash in China, and the employer provides them with lodging, clothing, bedding, mosquito-nets, food for the whole term of their agreement, and cash at the rate of one dollar *per mensem*. There may be other concessions, but these are essential. At the end of his term of service the sinkheh becomes a ‘*laukkeh*,’ or ‘old hand,’ at liberty either to work

for himself or to re-engage on the mine or plantation, which he generally does, on some profit-sharing basis.

In what follows it will be convenient to reserve the term '*laukheh*' for such recontracting coolies, whether engaged upon mines, estates, or in other employment, retaining that of 'free coolie' for those who work for themselves, and supply their own necessities.

The principal difference between *sinkhehs* and *laukhehs* is that the latter, having no longer, as when *sinkhehs*, a debt to work off, receive more wages. Part payment is usually taken in truck—food, clothes, tobacco, opium—or even shares in the mine itself, profits on which are realizable at each ensuing New Year, but part is always demanded and paid monthly in cash.

In small mines often, and nearly always in mines adjacent to villages where supplies can be easily obtained, the *laukhehs* purchase their individual necessities themselves. But in large concerns, and always in remoter mines, stores are got by the management from traders, to be resold to the coolies at enhanced 'customary' profit. Not seldom the mine manager and proprietor both are interested in the truck, the sales of which, if the mine is at all a paying one, it is to their advantage to make as large as possible. By this ancient system of sweating, the cash outgoings to coolies, kept heavily indebted for food, opium, etc., are reduced to the lowest margin.

It will now be seen how these facts affect the consumption of rice by the different classes, and so their tendency to acquire any disease which could be conveyed by it.

The *sinkheh* is, by his contract, fed for his term of service free of all charge (except his labour) by the employer. His food being thus paid for, as it were, in advance, and fully earned by daily hard work, it is an elementary appeal to his instincts to eat as much as possible. So far from any check being put upon this tendency, quite the reverse condition obtains. The employer encourages ample feeding, believing that it will produce better work. The *sinkheh's* rations are not measured nor stinted, but they are not varied. Rice (uncured, either Rangoon, or more generally Siamese), salt fish, salted egg, a few black beans, or sprouted pulse, or other grain, a little fluid curry-stuff, once a week a small portion of fat pork, comprise the list of comestibles commonly given. Of the whole bulk, rice forms, in the day's rations, 90 per cent.

The quantity of rice which a healthy coolie on a mine or

estate will consume in the course of a day is astonishing, and will be hardly be believed by those who have not, like the writer, observed them closely. Rice is eaten cold, or as hot *kanji* (gruel)—a large bowlful—on turning out for work in the morning at 5.30 a.m., again to the extent of 1 or 2 pounds weight cooked¹ on returning from it at eleven, this time with vegetables, etc., added. Another meal—a pound or two—is taken before going out to the afternoon's task at 1 p.m. At evening, the principal meal of the day, another pound or two of rice, with adjuncts, is taken about 5 or 6 p.m. Soon after sunset, if he does not frequent the gambling-table or theatres, the coolie retires to sleep, but during the night he gets up at odd moments to consume extra meals. On an average, the active mining coolie eats a quantity *weighed dry* of between 1½ and 2 pounds of rice daily. The customary allowance, Mr. J. C. Pasqual, chairman of the Selangor Miners Association, and himself an active manager and owner of mines in the Native States, informs me, is 45 'catties' a month, or almost exactly 2 pounds per diem. The allowance found sufficient for *prisoners* on hard labour in Singapore Prison from 1885-1897 was 14 ounces, but this was supplemented by bread, and other food. In most gaols now, 22 ounces is given.

In the Japanese navy and army beri-beri constantly occurred when as much as 26 ounces of rice was eaten with little else daily.

It is to be remembered that the coolie, who is thus simply gorged with rice as a sinkheh in Malaya, was previously a peasant earning, perhaps, by laborious tillage of patrimonial fields no more than a single dollar a month in China, and able to buy but just enough rice for bare subsistence, some 3 or 4 ounces forming his daily portion.

The sinkhehs, then, are an example of *rice-eaters in excess*; they are those who eat rice in greatest quantity both absolutely and relatively. It is among them, accordingly, that it is to be expected the incidence of beri-beri should be the greatest, and its course most fatal.

In the laukheh the habits of the sinkheh still remain. He devours with the same greed, still eats rice in great excess. But the laukheh, as we have seen, receives higher wages, and is paid part cash, and thus he is in a position to buy what food he chooses.

The result is that he largely supplements his rice by other

¹ Rice will absorb at least 70 per cent. of its own weight of water.

articles of diet. All his spare cash which does not go for opium or gambling is spent on his belly. He riots in duck, and durians, and fat pork, in sweets, in shell fish, and all sorts of eatables. Hence, in the diet of the laukheh, the proportion of rice is always relatively less than in that of the sinkheh, and, since the extras must replace a certain amount of the staple, to that extent nearly always less absolutely also. So far as the tendency to get beri-beri depends on quantity of rice eaten, therefore, the disease should be less prevalent among laukhehs than sinkhehs.

Last to be considered among the natural divisions of the coolie population which have been indicated is that of the free coolies. These, for the most part, have in their time served as sinkhehs or laukhehs, and subsequently taken up other trades. They are found as independent miners, gardeners, rice-planters, petty shopkeepers, boatmen, rickshaw-pullers, mechanics or 'tukangs,' and labourers of all kinds.¹ As before, a line of relative wealth marks the only distinction between these individuals. Those who succeed and make money spend it freely in food, and procuring everything eatable which they desire, eat *relatively* less rice than the sinkheh, however much the absolute quantity may be. Others, who fail to make money, remaining always poor coolies, have hard work to get rice enough.

If circumstances encourage the sinkheh and laukheh to eat rice to excess, quite the reverse conditions apply to coolies of this class. For such men, earning as they often do but scanty living with hard toil, it is frequently a matter of difficulty to gain rice enough.² Whereas none of the others are settled, or have wives or families, many of these have both. These circumstances tend to frugal living, while often a desire to save enough to enable them to return to China leads them to economize even at the expense of their diet.

In this class of the employed the quantity of rice consumed is least of all, and it is among them therefore that the incidence of beri-beri should be least severe.

Each of the inferences here made as to the relative incidence of beri-beri upon the different classes of coolies described is justified in experience.

The fact has impressed itself not only upon the writer, but upon all those who have studied beri-beri in Malaya, that it is

¹ Mechanics are called *tukangs* by the Malays.

² Those who are better off, making a good living, and able to indulge in a varied diet, may be reckoned with the wealthy, the 'towkay' class.

the sinkhehs who are, at every place where the disease occurs—or, in other words, in every quarter into which they are introduced—most severely affected. Next to them, the laukhehs, whether on mines or elsewhere, suffer.

The least afflicted are the free coolies, of whatever occupation.

It is to be regretted that no body of figures has been brought together to illustrate these conclusions statistically. The very notoriety of the facts, it is, which has seemed to render such a task superfluous. But for the benefit of those unacquainted with the circumstances at first hand, some citations may be made from another observer, which have the advantage of being derived from a hostile witness, to confirm the statements of the present writer.

H. Wright, having noticed the division of the Chinese into classes such as the writer has described, states that it is in the class of sinkhehs that 'the largest number of cases of beri-beri develops' ('Study,' par. 144). 'The great majority of Chinese who contract beri-beri are sinkhehs, although sinkhehs are not the more numerous class in the whole population. . . .' (par. 158). Referring to laukhehs, 'who drop tin-mining and take to trade as shopmen or hawkers, or as shop hands,' he says: 'Amongst this class beri-beri is rare, as is also the case with Chinese engaged in domestic service. . . . Beri-beri is rare likewise amongst rickshaw pullers.'

He gives an instance of the severity of the malady as attacking sinkhehs. 'Bentong, in Pahang, was notorious as a beri-beri centre. There, from October, 1898, to November, 1900, were some 800 *deaths* reported as due to beri-beri out of a total of 2,400 sinkhehs sent in' (par. 158).

In 1900 the *case-mortality* among affected sinkhehs at Bentong was reported by Dr. J. D. Gimlette as 799 per 1,000.

'No sinkhehs were sent to the mine,' Wright goes on to say, 'after April, 1901. The old *kongsies*¹ were burnt down, and new ones built, and the general hygienic conditions improved. Beri-beri began to die out, and on my last visit, in 1902, the state of health was in violent contrast to my first visit in 1901.'

Although a late addition to the material which has served in this paper for a discussion of the etiology of beri-beri, comment may be made upon the inconsistency of the conclusions as to its nature expressed or implied in these paragraphs of his 'Study' by Wright.

¹ Miners' dwellings.

In par. 159 he refers to Bentong as a 'notorious beri-beri centre,' which implies, if anything, that it was the *place* to which the production of the disease was due, a view which he emphasizes later in his paper. In par. 191 he states that the sinkhehs newly imported to Bentong were 'thrown into association with "infected" laukhehs,' an implication that the disease was derived from *personal contagion*. Finally, it is to 'general hygienic' improvement that the dying out of the epidemic is attributed.

In all this, the point of real importance is missed. It is not directly stated, but is left to be inferred from Wright's account—and the writer is able to assert as much from knowledge of the facts gained by himself personally at Bentong, both with regard to this epidemic and earlier ones—that the laukhehs were, at Bentong as elsewhere, less affected by beri-beri than were the sinkhehs. When, following 1901, *no more sinkhehs were sent to the mines* at Bentong, and beri-beri began to die out, it was to the first of these two facts that the last was due. The disease began to die out, not because there was any change in the place, or in hypothetical emanations from, or of infective 'foci' in the place, but because there was *no more material*, there were no more sinkhehs, to provide the cases.

Bentong affords but one among hundreds of such instances, where history repeats itself in the production of similar epidemics, and the expression of similar fallacies of observation.

The principal circumstances attending the opening up of one new mine, or mining district, or even of an estate, are common to all. Old hands are already employed for the most part at old mines. For new ventures fresh hands—sinkhehs—must be engaged. The conditions already described produce beri-beri among the sinkhehs. An epidemic rages for the initial years of the mine's working—those for which the sinkhehs are engaged as such. It ceases when the ground has become 'more opened up'—the time at which, their term of contract at an end, the sinkhehs become laukhehs, the conditions applying to whom render them, as explained, less liable to beri-beri.

To cite some further instances from Wright. In par. 190: 'In the older mines . . . beri-beri is rarer. Most of the coolies in these mines are laukhehs. . . . A few sinkhehs are introduced each year . . . and it is generally among this class that acute pernicious beri-beri appears.' Par. 193: 'At the Tronoh Valley Mine . . . no beri-beri has been known for some years past, except one bad outbreak in 1901. This occurred in a kongsi

where were herded together a large number of surface-working sinkhehs. It was altogether confined to them, none of the laukhehs contracting the disease.' The laukhehs would be equally 'herded together,' and the site of employment might be identical.

The comparative freedom of the laukheh from beri-beri, as compared with the sinkheh working beside him in the same mines, which is an undoubted fact, is also one of which it is difficult to conceive any possible explanation other than that given, which harmonizes with all else that is known of beri-beric epidemiology. But the truth of that explanation is made still more apparent by another observation of Wright's which the writer can also confirm.

It is that, when deprived of the advantage which he usually has over the sinkheh of getting better and more varied food, the liability of the laukheh to beri-beri equals the sinkheh's; this happens in prisons.

In an analysis of 90 cases originating in Pudoh Gaol under his own observation, H. Wright shows that almost exactly half (44) who were not miners were laukhehs, while of the remainder (46) who were mining coolies most were laukhehs.

Experience justifies, then, the deductions made. Among the Chinese—the greatest sufferers from beri-beri—the *incidence of the disease is proportionate to the quantity of rice eaten*. Sinkhehs are most, laukhehs next, and free coolies are least prone to acquire the disorder.

2. **Evidence from Manila.**—One of the most striking examples ever recorded of the effect of a *relative* increase of quantity of rice eaten in determining the occurrence of beri-beri was perhaps the outbreak in *Manila*, the description of which by Königer has already been quoted. The natives, though always rice-eaters, consuming fresh rice, had never suffered from beri-beri before 1882.¹ But in that year the inhabitants of Manila and the neighbourhood, suddenly deprived, through the operation of several rapidly succeeding disasters—cholera, a cyclone, and a great flood—of all the usual adjuncts—fish, fruit, vegetables (yams)—by which their simple rice diet was wont to be supplemented, were reduced during two months or more to subsistence on rice *alone*. This may have been the customary fresh rice, or, and

¹ So Königer. But Fiebig (*Gen. Tijd. v. N.I.*, xxx., p. 438) says that Schneider (*ibid.*, xxiii. 4) had treated beri-berics at Sambuangan in the Philippines as early as 1852.

more likely, a bought, stale, uncured variety, but the fact of importance is that *only rice* was eaten.

Beri-beri then appeared for the first time in the history of the island, and continued to be severe upon all the rice-eaters until the normal conditions were restored.

Similar circumstances recurring in 1903 produced a similar epidemic once again in Manila among troops employed by the United States Government. This observation made by Smart has already been referred to (p. 102).

Other illustrations of the dependence of beri-beri on proportion of rice eaten are to be found in the history of public bodies or institutions.

3. **Evidence from Japanese Army, Fleet, and Prisons.**¹—Foremost among these is the oft-cited instance of the Japanese fleet, army, and prisons.

Beri-beri, which had long been a severe scourge of the sailors, invaliding no less than 30 per cent. of them annually, declined, to disappear entirely, within a year after the quantity of rice in the daily ration had been reduced by 5½ ounces, and from 56 to 32 per cent. of the whole.² For the rice withdrawn, fish, meat, flour, milk, and other articles were substituted. The reduction in the absolute quantity of rice eaten was made relatively even more effective by this addition of more nutritious components to the dietary.

Takaki,³ who introduced the reform, under the belief that beri-beri was essentially due to nitrogen starvation, was persuaded that the disappearance of the malady was directly due to the change. Saneyoshi,⁴ Director of the Naval Medical Service of Japan, reviewing the whole question later, in 1898, states that the same measure, applied to the army and the prisons, in all of which the disease had prevailed previously, led to the same results. He states his conviction that it was to the alteration in the diets that the 'extirpation' of the disease in both the army and navy and prisons was due, and adds 'that no other hygienic improvement has been recognised as having anything to do with this result.'

The improvement in the naval rations effected by Takaki had

¹ See 'Examen des mesures prises contre la kakké dans la Marine Japonaise.' *Arch. de Méd.*, November, 1893, vol. lxx., p. 233.

² From 782 grammes (26½ ounces) to 648 grammes (22½ ounces).

³ *Sei-i-Kwai*, August, 1885; April, 1886; April-May, 1887.

⁴ *Ibid.*, April-May, 1901 (vol. xx., 415).

the effect of increasing the nitrogen proportion from 1 in 28 to 1 in 20, which was still, he remarks, less than desirable. The absolute quantity of rice eaten was reduced from 782 grammes (27.7 oz.) to 648 grammes (22 oz.), while the auxiliary articles were increased from 607 to 1,235 grammes daily.

The effect was enormous.

Prior to 1884, when the new diet was introduced, there had occurred on an average 1,586 cases of beri-beri in an average force of 4,886 men—a ratio of 325 cases per 1,000—with 41 deaths *annually*. In the year 1884, when the changed diet began to come into operation, and the following years, with a steadily-increasing force the cases dropped to 718, 41, and 3 annually, the case-mortality from 3.96 per cent. in 1883 to 1.11 in 1884, and nil afterwards. The following figures are given by Takaki :

Year.	Strength of Force.	Number of Cases of Beri-beri.	Admissions to Hospital for Beri-beri.	Deaths from Beri-beri.	Case-mortality per Cent. in Hospital.	
Old diet scale {	1881	4,641	1,163	300	30	2.58
	1882	4,769	1,929	545	51	2.64
	1883	5,346	1,236	378	49	3.96
New diet scale {	1884	5,638	718	209	8	1.11
	1885	6,918	41	25	nil	nil
	1886	8,475	3	nil	nil	nil

The alteration in change of diet was followed by extraordinary diminution in the sickness-rate, not only from beri-beri, but all other, except genito-urinary, disorders also. This has been held to imply that the reduction of beri-beri was the result less of withdrawing its specific cause than of general improvement in hygiene, sanitation, etc. But assuming beri-beri to be a slow process of poisoning through food, it is obvious that a gradual depravation of the system must long precede that sudden break-up of the nervous organization which forms the condition known as beri-beri, and that the liability of the subjects of such a depraved state of health to the attacks of other disorders must be greatly increased. Removal of the cause producing beri-beri would, on this showing, naturally lead to the reduction of sickness from all causes. A similar relation of beri-beri to other sickness in the Malay States has been pointed out in the opening of this work. The influence in this direction may aptly be compared with that of alcoholism.

Among naval prisoners, who in 1883 had a diet containing only 216 grammes of nitrogen to 7,059 of carbon (1 to 32), the

beri-beric sick-rate was 61 per cent. (68 cases among 113 men). In 1885, a year after the nitrogen had been increased to 289 grammes, and the carbon reduced to 5,839 (1 to 20), there were *no cases* among 168 prisoners.

4. **On Training-ships.**—Takaki gives results of experimental alterations of diets in relation to beri-beri on two training-ships, which afford proof of its dependence on rice equally convincing as the naval reform.

On the *Riujo*, carrying 276 men and officers, there occurred, during a cruise of 272 days from Japan via New Zealand, Valparaiso, and Honolulu homeward, 169 cases of beri-beri, with 25 deaths; 160 of these were among the sailors and petty officers, 9 among the officers. These cases happened before reaching Honolulu. The diet then contained a proportion of nitrogen to carbon rising from 1 to 28 (sailors) to 1 to 20 (officers). The experiment was made of increasing the proportion of proteid in the rations, so that the nitrogen should be raised to 1 to 16 (sailors) and 1 to 11 (officers). On this diet no cases occurred. Takaki says 'careful examination following the course of the voyage with regard to the appearance and disappearance of beri-beri shows that when the experimental quantity of nitrogen substituted was diminished, and the carbohydrate increased in quantity, it (beri-beri) always appeared. On the contrary, when the reverse was the case, it retrogressed or disappeared entirely.'

On the *Tsukaba*, sent out to follow the same course as the *Riujo*, it was arranged that the rations should contain nitrogen and carbon in the proportion of 1 to 15. But the two elements were actually taken by the men in the proportion of 1 to 17 (total nitrogen 450 grammes, total carbon 7,650 grammes). 'In the course of the 287 days of voyage 4 cadets and 10 men were attacked by the disease. It appears that the cadets were unable to take condensed milk, of which their fellows took 1 pound weekly; also 8 of the 10 men had been unable to eat meat as the others did.'

The carbohydrate element in all these instances was rice. The relative increase or decrease of nitrogen to carbon, which Takaki clearly shows beri-beri to have followed, was thus also an index of relative increase or decrease in the quantity of rice consumed.

In regard to the dietetic theory of beri-beri, the reflection arises that deprivation of nitrogenous food is one thing, excess of carbohydrates another. The fallacy attends the nitrogen-starvation theory that the presence of nitrogen-starvation is everywhere, in Takaki's instances, complicated with not only

relative, but absolute, *excess* of carbon. It does not follow—has not, indeed, been anywhere observed—that the effect of a demonstrated lack of proteids should be rendered any worse by the presence of excess of carbohydrates. Moreover, while the results of deficiency of nitrogenous stuff present the familiar picture of simple starvation, the features of beri-beri are those of distinct poisoning. Now, it has never been shown that excess of any pure carbohydrate produces such a result. The presumption is, then, that where this event does occur, it happens, not because carbohydrates in excess are dietetically toxic *per se*, but because the particular kind of them employed is poisonous.

Moreover, Takaki's view that the disease is essentially the result of nitrogen-starvation conflicts with general evidence, and is, indeed, hardly justified by the data upon which he founded it. If it be true that fluctuations in beri-beri have everywhere coincided with similar increases or decreases of nitrogen-starvation in Japan, the same is not true elsewhere. Nor, indeed, in Japan is nitrogen-starvation the *only* factor, changes in which have exactly coincided with those of beri-beri. Since in all cases the principal staple of diet was *rice*, wherever there was nitrogen-starvation it meant that excess of rice, absolute or relative, was eaten, and when this rice was toxic the result was beri-beri.

Saneyoshi clearly summarizes the Japanese experiences in the *Sei-i-kwai Medical Journal* for April and May, 1901, and adds many interesting observations to those recorded earlier by Takaki. It appears from his account that prior to 1884 and after, the average diet of Japanese sailors and of soldiers in barracks was as follows :

Diet.	1883.	1884.
	Grammes.	Grammes.
Rice - - - - -	782	648 ¹
Fish - - - - -	96	150 ²
Meat - - - - -	73	300
Vegetables (fresh) - -	145	450
Vegetables (pickled) -	215	75
Sugar - - - - -	18	75
Miso (beans) - - - -	60	50
Milk - - - - -	—	45
Flour - - - - -	—	75
Beans - - - - -	—	15
Total -	1,389	1,883

¹ Or bread 600 grammes.

² Or biscuit 490 grammes.

228 THE CAUSE AND PREVENTION OF BERI-BERI

To the 1884 diet 'were to be added' also *shoyu* (pea-sauce), 60 grms. ; fat, 15 grms. ; salt, tea, and vinegar, each 8 grms. ; *sake* (rice-spirit), 90 grms.—making a total ration of 2,066 grms.

		Proteid.	Fat.	Carbohydrate.
		Grammes.	Grammes.	Grammes.
The old diet contained	-	109·29	158·9	622·32
The new diet contained	-	196	43	775

Without contesting that the first rations were inferior, or the latter a great improvement, can it be contended that there was serious nitrogen-starvation on the old diet? Or if there was, would beri-beri result from it? The answer is that there are thousands of natives of the West, European operatives, and the poor generally, who suffer continually from a far more serious want of nitrogen, and labour hard withal, who yet do not get beri-beri. The following table, in which various diets in use in the old world are compared with the Japanese regulation naval diets, will demonstrate this clearly :

Composition of Various Diets (exclusive of Salts and Water).

	Proteid.	Fat.	Carbohydrate.	Authority.
	Grammes.	Grammes.	Grammes.	
Barely sufficient to sustain life	75	40	325	Richards
Low-fed operatives in Eng-	91·2	19·2	635·4	Playfair
land - - - - -	(3·22 oz.)	(0·64 oz.)	(22·43 oz.)	
Standard sufficient diet with-				
out work :				
Females - - - -	80	80	300	Atwater
Males - - - - -	100	100	360	do.
Japanese navy regulation				
diet (old) - - - -	109·29	158·9	622·32	Saneyoshi
Physiological standard on				
light exercise - - -	118	50	500	Voit
Standard diet on hard work -	120	35	540	Atwater
Well-fed workman - - -	130	84	404	Moleschot
Physiological standard on				
hard work - - - - -	145	100	500	Voit
Well-fed operatives in Eng-	174	72·6	558	Playfair
land - - - - -	(5·8 oz.)	(2·4 oz.)	(18·6 oz.)	
Japanese navy regulation				
diet (new) - - - -	196	45	775	Saneyoshi

If there was nitrogen-starvation in the earlier diet, it was not, then, excessive, while even greater lack of proteid is endured by many classes who have to work, without beri-beri resulting.

Still, the abolition of beri-beri on the adoption of the more

nitrogenized rations became a fact, and remains one. Its explanation lies in the *quantity of rice* consumed, which previously was greater, and later was less absolutely, and by reason of the very great increase of non-carbohydrate elements in the new diets became relatively even much more reduced.

Of the early ration the total weight was 1,389 grms., of which rice composed 782 grms., or 56 per cent. The later diet amounted to 1,883 grms., of which rice was only 648 grms., or 32 per cent., of the whole. The old diet contained absolutely 702 grms. (30 per cent.) more rice than the new, and the actual weight of dry rice consumed (26.8 ounces) must be deemed excessive.

In spite of the wide notice which the Japanese results have attracted, their importance, in the writer's opinion, has not even yet been fully realized.

The following table from Saneyoshi illustrates the great prevalence of beri-beri before the alteration in diets, its remarkable suppression by that measure, and the maintenance of that condition of exemption down to a late date.

Prevalence of Kakké (beri-beri) in Japanese Navy.

Year.	Average Strength of Force.	Cases of Kakké.	Percentage of Force affected.
Old diet.	1878	1,485	32.8
	1879	1,978	38.9
	1880	1,725	34.8
	1881	1,163	25.0
	1882	1,929	40.4
	1883	1,236	23.1
	Average	1,586	32.45
	1884 ¹	718	12.74
	1885	41	0.59
	1886	3	0.04
New diet.	1887	—	—
	1888	—	—
	1889	3	0.03
	1890	4	0.04
	1891	1	0.01
	1892	3	0.03
	1893	1	0.01
	1894	29	0.26
	1895	17	0.13
	1896	11	0.08
	1897	22	0.14
	1898	16	0.08
	Average	10	0.07

¹ New diet introduced. The change ordered, but not brought generally into effect until part of the year was over.

² See note on next page.

In the year when the dietary change was introduced 'no other sanitary change worth mentioning,' Saneyoshi states, 'was made.' Such measures as had been taken were attended with no beneficial effect. It was not until after the introduction of the new diet system that any result was seen.

It is interesting to note from this table that though the cases were absolutely few, compared with former days, there was relatively a great increase about 1894 and onwards—a period at which exacerbations of beri-beri happened in many other places.¹

5. Rice Reduced in the Prisons Originally for Economic Reasons.—The beneficial effect of improved dietary was not confined to the navy. It had, indeed, first been demonstrated in the prisons, where, Saneyoshi says, barley was substituted largely for rice originally as an economic not a hygienic measure. 'In the prisons of the Empire for many years,' he states, 'the allowance of food assigned to a prisoner was 750 grammes of rice, and some poor auxiliary food costing 1 or 1½ cents, and *there were numerous cases of kakké*. In 1875 prisoners were allowed to have rice and barley, subsequently in 1881 it was enacted that in every prison barley and rice should in the proportion of 6 to 4 be supplied as the chief food.

'In the prisons where barley was used from an earlier date kakké had become much reduced, and since barley was made to form a part of the regulation food it has *become very rare in every prison*. This change of diet in prisons was originally proposed with a view to economy. It was quite an unexpected result that the change turned out to be the means of bringing the disease under control.'

6. The Change in the Army.—'Again, in the army the case was similar. Formerly, in the barracks of every district many cases of kakké invariably occurred in every year, and the difference in number seemed to depend on the prices of commodities in the respective districts.' (When rice was dear the cheaper barley was more used.)

'In the army the daily ration of every soldier was, in former days, 900 grammes (30 ounces) of rice, and auxiliary food costing 6 cents. Accordingly, when the price of daily necessities was cheap, a comparatively larger quantity and better quality of food could be obtained, while the contrary was the case in such

¹ These were also the years during and after the war with China. Scarcity of barley and other exigencies then increased the relative amount of rice eaten. See Tabusaburo Yabé, *Arch. de Méd.*, November-January, 1900.

cities as Tokyo and Osaka when the price was very high.' (And in these beri-beri was proportionately prevalent.)

Saneyoshi gives a list of twenty such places in which the proportion of beri-berics ranged from nil to 50 per cent. of the garrison during the years 1882-1884.

The cases occurring in the whole army during these years were as follows :

Year.	Force.	Cases of Beri-beri.	Proportion per Cent.
1882	39,975	7,966	19.9
1883	38,717	7,128	18.4
1884	36,483	9,643	26.7

'The fact that the introduction of barley into the prison diet had almost exterminated kakké was suggested to the military authorities, who accordingly tried the scheme, with the result that the disease began in 1885 to decrease, and at last the ratio of cases *per thousand of force was reduced to less than ten.*

'Subsequently, barley was used in barracks at various places, and at the present day it is universally used in the army, and kakké cases have become rare.'

It will be observed that the decline of beri-beri in the prisons began and continued (1875-1883) while the disease was still 'raging' in the navy and army ; that the adoption of the change in 1884, although not then made general, reduced the prevalence in the navy by one half (from 23 to 12 per cent.), *while the disease remained unchecked in the army* (26 per cent.). All this time beri-beri was, and has since remained, just as prevalent everywhere throughout the country wherever it had previously been so, while the exemption of the army, navy, and prisons from it has continued.

The *post hoc propter* argument which has been urged against the validity of the inference drawn from the Japanese experiences cannot, therefore, be maintained. The statement of Baelz, for instance, which Scheube quotes with approval,¹ that *beri-beri disappeared in all the barracks* simultaneously with its 'extermination' from army and navy, although, as he says, the improved diet was purposely not adopted in all of them simultaneously, is completely disposed of by the figures given by Saneyoshi. For the disease was stamped out in neither navy nor army in any one year,

¹ 'Krankheiten der Warmen Länder,' 2nd edition, Jena, 1900.

while the reform which led to this result was adopted in the navy in one year and the army in another. With which event, then, was the 'disappearance' of the disease from the barracks, according to Baelz, 'simultaneous'? It is evident that much latitude is given to the meaning of the word, so that it may very well be that the real coincidence was between the disappearance of beri-beri from the barracks and the adoption, which Saneyoshi expressly states took place later, of the use of barley in them also.

In Japan beri-beri is usually most prevalent in the summer, and Saneyoshi attributes this fact to the greater consumption of rice by lower classes at that season.

'In the summer,' he says, 'fish are much used among us, and as they are liable to putrefaction if not eaten at once, they become so dear that ordinary people cannot afford to obtain fresh fish. The Japanese, therefore, often contenting themselves with so-called *chazuki-meshi* (a plain-boiled rice mixed with tea), and thus, owing to the complete insufficiency of other nutritious foods, they become more liable to attacks of kakké.'

7. Exceptional Epidemy in Prison at Nijgata.—A singular outbreak of beri-beri occurring at a Japanese prison after the regulation new diet which had been supposed to prevent its occurrence had long been in force everywhere afforded an illustration of the exception which 'proves the rule.'

'In January, 1899, kakké suddenly broke out and raged at the prison of *Nijgata*, and from January 15 to the end of March about 400 persons out of some 1,000 prisoners were admitted to hospital. This was apparently a very strange phenomenon now that kakké had become a rare disease in prisons, since the introduction of the barley and rice diet, and investigation was made by despatching a committee from the Navy Medical Department to the said prison. As a result of the investigation, it was found that, on account of the dearth in that region, and consequent rise in the price of barley and rice, *cheap rice imported from China* had been bought since December of the previous year, and used in place of the native barley and rice. At the same time, it was found that the disease attacked in greater number the prisoners who were supplied with *white* Chinese rice, while it affected in smaller number those who had red Chinese rice. The red and white rice differ in that the red, compared with the white rice, has a larger amount of fat and albumen. . . . At the said Nijgata Prison from March, 1899, barley and rice were used again, which resulted in the gradual disappearance of kakké.'

'At Nijgata, in January, the temperature is at its lowest, and just at that time the ground was covered with snow several feet deep.'¹

To summarize: In the foregoing instances comparison is made of the incidence of the disease upon those eating more and others, similarly circumstanced, eating *less* rice.

(a) It was practically equally prevalent at one time in prisons, army, and navy. Reduction of rice in the prison fare (beginning by permission in 1875 and by regulation in 1881) was followed by decrease of prevalence of beri-beri, the same rate in army and navy continuing.

(b) Reduction of rice in the navy diets in 1884 was followed by decreased prevalence in the navy, that in the army remaining the same.

(c) Reduction of rice in the army rations in 1885 was followed by decreased beri-beri in the army also, its prevalence in the civil population throughout the country remaining the same.

In all these institutions the decrease of prevalence followed *immediately* upon the introduction of the new diet, *the reduction in quantity of rice eaten*. The effect was observed not at one place, but in very many different places, different ships, different garrisons, different prisons, scattered all over sea and country, wholly unconnected with each other, *simultaneously* with the adoption of the measure in them.

Moreover, in each instance, there was not mere 'decrease of prevalence' of the disease: it vanished.

In the navy, instead of some 30 *per cent.* of the force annually sick with beri-beri, as had been the case prior to 1884, the rate was reduced to 7 in 10,000.

Where *over 10,000 actual cases* had occurred year by year in the two services, *now only a score* or so were seen.

These few cases sporadically happening serve to show, however, that the agent of the disease is still abroad in Japan, as, indeed, the civil hospitals abundantly prove.

8. **Outbreaks among Japanese Colonists.**—Saneyoshi relates,² among other instances, the following example of epidemics of beri-beri which nothing but excessive rice-eating could have produced.

'The island of Shimushu, at the northern extremity of the Kurile Islands, faces Kamschatka, and washed as it is by the cold current of Behring Straits, is the coldest region in our Empire. Up to 1884 some 100 aborigines lived on the island,

¹ Saneyoshi, *loc. cit.*

² *Loc. cit.*

but in that year were all removed. For several years Shimushu remained deserted. In 1896 six persons seeking to colonize the place passed the winter there as an experiment. In the coldest month, January, four were attacked by kakké, and three died. According to information furnished by the survivors, the only food they had left was *rice, miso (beans), and salt*.

'In the summer of 1896 37 men and 11 women went to the same island, and at the end of November kakké began to appear. Among the men there were 26 cases, 6 fatal. Among the women 9, of whom 1 died. Dr. Ito, who accompanied the party, stated that, as the cold increased, hunting and fishing stopped, and no fresh meat could be obtained—cattle taken with them died of the cold. While during the summer months, being actively employed, their appetites were good, they could eat also tinned provisions. In winter, when there was no exercise, their appetites failed, and they revolted at these sorts of provisions, to which Japanese are not habituated. They reverted, therefore, to the rice and pickled vegetables, which were accustomed articles of diet.'

In 1897 a larger number of emigrants went out, and taking care to organize active exercise during the winter months, managed to consume a proper proportion of meat, etc., in tinned stores, and they all kept well.

Saneyoshi, who ably reviews the relation of rice to beri-beri in 1901, it is true, still adheres to the view of Takaki, that the disease is primarily a nutritive disturbance. 'Beri-beri and rice,' he says, 'are inseparably connected. *Lack of nutritive substance is the cause of beri-beri.*' It must be repeated that this view, debatable on the Japanese statistics, is wholly negatived by the fact, demonstrated often enough, that the disease may, and does, attack those in whose rations there is no deficiency of nitrogen, nor any other dietetic want. Wright's experiment at Pudo Gaol proved this.

But if these observers have erroneously interpreted the *mode* in which rice operates to produce disease, their evidence as to the *fact* of the dependence of beri-beri everywhere upon rice, in whatever manner the effect may be produced, is not therefore of less, remains, indeed, of monumental importance.

The extirpation of beri-beri from each one of the many vessels, the various regiments, the several garrisons, the prisons, in which it was prevalent in Japan, in each case at different intervals, but simultaneously with reduction in the amount of rice eaten, is not a single isolated fact, but a whole body of instances, such as

it is impossible to attribute to coincidence, or to regard as due to any other cause than the one authenticated factor—the change of diet—which was new and common to all. It is idle to attribute to vague ‘general improvements in hygiene’ or ‘sanitation,’ no single principle or resource of which has ever in the slightest degree modified the course of the disease in other places—gaols, ships, and asylums—where such measures have been applied, an abolition of disease so complete as that of which we have here an example. It is impossible to believe of the disappearance of beri-beri from Japanese navy, army, and prisons, otherwise than with Takaki and Saneyoshi, that it was directly due to the alteration of diet. In that alteration the essential, as we have seen, was not the adjustment of the physiological requirements, not the elimination of rice from the diet altogether, but merely a *reduction in the quantity* eaten daily.

Saneyoshi also refers to the well-known New Caledonia outbreaks, which were described by Grall, Porée, and Vincent,¹ who ascribed them to decomposed fish, but which he considers were due to lack of sufficient nitrogenous food.

9. **The New Caledonia Epidemy among ‘Annamese.’**—In February, 1891, a party of 758 healthy Annamese left their own country to colonize these islands. They had arrived but a month at Orphelinat when beri-beri broke out. They removed to Freycinet, but the disease continued to extend. Again to Kautio; there were more cases still. In all there were several hundred cases and seventy deaths.

The rations of these immigrants then consisted of 1 kilo (35 ounces) of rice, 200 grammes (6.3 ounces) of salt fish, and nothing else.

The fish was actually putrid, but was yet used until April, when the whole stock was thrown away, and new rations were issued. These consisted of 1 kilo rice as before, 200 grammes of meat, 100 grammes vegetables, but of the latter there was no proper supply. Little or no difference in the prevalence of the disease ensued.

At the *end of July*, for the first time, the immigrants were put in a position to buy their own food.

By *August* the disease disappeared.

Hagen² offers certain observations of his own made in New

¹ *Arch. de Méd. Nav.*, t. lxiii., Nos. 2, 3, 4, February-April, 1895.

² Hagen, ‘Du béri-béri à la Nouvelle Calédonie, et de quelques observations tendant à prouver son caractère contagieux.’—*Revue Médicale de l’Est*, t. xxv., No. 2, January 15, 1893, p. 42.

epidemics is raised by Hagen's account of them. His cases, as he records them, are far from corresponding to the type of true beri-beri—of the complaint, at least, which abounds in British and Dutch Malaya. For, of the latter affection, in every epidemic where there are serious or severe cases, some at least will show nervous disorder in definite and marked signs, paræsthesia, anæsthesia, motor paresis or paralysis, tenderness of nerve-trunks and of muscles, hypermyotonus, followed by loss of reflexes, and above all, early and marked muscular atrophy. There will also certainly be early diminution, followed later in recovering cases by increase of urine. Nor will it be possible to find patients gravely ill in whom it can be said that the heart remains normal, as it is definitely stated by Hagen to have been in his cases. The picture he presents resembles the disorder with which beri-beri has been so frequently confounded—epidemic œdema, and however valid his conclusions may be as regards the possible contagiousness of this disease they afford no evidence whatever of this characteristic attaching to beri-beri.

10. **New Caledonia Epidemy among Japanese.**—A second party, of 600 healthy Japanese, went to the same islands, arriving after an eighteen days' voyage in January, 1892. They settled at two places, at both of which beri-beri broke out in the middle of March, and by the end of May there were 438 cases. The diet of these men was composed of 900 grammes (31 ounces) of rice, and 50 grammes (2 ounces) fish, but the fish proving dear, was replaced by 110 grammes of dried shrimps, seaweed, cabbage, or pickled plums.

On substituting for the former rations 225 grammes of fresh meat and 1,500 grammes of bread the disease disappeared.

Absolutely, in both these instances, the quantity of rice eaten daily was very large. Relatively it was, in the periods preceding and during the outbreaks, enormous. In both we have the cause of the disease reduced in the clearest possible way to food.

The victims left their own country well,¹ and were healthy on arrival. In the case of the Annamese, the country they came from was one in which, in spite of the Chantabun epidemy, the disease is not endemic. In the country they came to the disease had never before been known, the inhabitants (Polynesians) subsisting less on rice than on taro, fish, etc.

¹ The Annamese had no previous acquaintance with such a malady. The 'cursed island' to which they had been brought was bewitched, they said. The lamentable record given by Vincent, Porée, and Grall, shows a state of affairs that might well have struck any race with panic.

Only two articles were consumed, of which one (fish) has been proved conclusively by later experiments to play no part in the causation of beri-beri.¹

There remains rice, therefore, as the only possible source of the disorder.

Change of locality, it is worth pointing out, was in these instances without effect, since no alteration in diet was secured by it. But when, among the Annamese, the proportion of rice eaten was made less, by their obtaining for themselves a more varied diet, and when, with the Japanese, a bread diet was substituted for the rice, the disease promptly ceased.

II. Second New Caledonia Epidemy among Japanese.—A decade later this disastrous history was repeated in the same place. I translate the following extract given by Firket (*Arch. f. Schiffs. u. Tropen. Hyg.*, Bd. IX., H. 2, February, 1905, p. 77) of an account published by Judet de la Combe of a second epidemy of beri-beri among Japanese immigrants into New Caledonia in 1900-1901:²

'The labourers were mainly Japanese recruited by an emigration agency, to the number of about 1,000. In spite of a medical inspection, probably perfunctory, undergone at the port of embarkation, many of these men were constitutionally liable to disease. Their lodgings were healthy. The rations, fixed by contract, could only be altered by mutual agreement between employers and employed, and consisted essentially of

Japanese rice	1,000 grammes
Fresh meat	250 "
Salt fish	90 "
Vegetables, dried	70 "
Vegetables, fresh	250 "

—with fat, tea, salt, and various Japanese condiments. The rice, specially imported for these labourers, was of very fine quality, and expensive.

'In spite of these favourable conditions, beri-beri appeared among the Japanese from the time of their arrival, and from September 1, 1900, to August 1, 1901—that is to say, during eleven months, there were 748, nearly three-fourths of the whole contingent, sick in hospital with beri-beri.

¹ At the Pudooh Gaol, Selangor, where, during an eleven months' observation by H. Wright, no fish of any kind was admitted, and yet beri-beri continued to occur.

² 'Morbidity et Mortality d'un convoi d'immigrants Japonais en Nouvelle Calédonie en 1901-1903.'—*Ann. d'Hyg. et de Méd. Col.*, t. vii., p. 326.

'Notwithstanding the opposition of the Japanese, who stuck to their rice, Dr. Judet de la Combe succeeded in substituting for 500 grammes of rice an equal weight of bread, and over and above the rations were added 50 grammes of meat and 20 of fat. This change was introduced in July 21, 1901,¹ and from August 1, 1901, to July 1, 1902—that is to say, during a second period of eleven months—there were only 28 cases of beri-beri in spite of the well-known tendency of this disease to recur. We draw special attention to the fact that it was the rations alone that had been changed, the other hygienic conditions remained the same.'

12. **Evidence from the Dutch East Indian Navy.**—Scarcely less remarkable than the Japanese experiences are the results which were obtained by similar measures taken at an earlier date in the Dutch East Indian Navy.

I condense the following from the very plain account of them given by Van Leent in 1880:²

The dietary of native crews serving in the Dutch Fleet in Eastern waters consists chiefly of rice, which, prior to 1874, formed 77 per cent. of the whole ration, the allowance being 1 kilo of rice with 300 grammes of meat (flesh), or fish, fresh, dried, or salted, as circumstances allowed.

The nutritive value of the ration was as follows :

	Grammes.	Proteid.	Fat.	Carbohydrate.
Rice	1,000	75	—	800
Flesh	300	54	20	—
Total	1,300	129	20	800

Also salt, vinegar, pepper, sugar, coffee.

¹ 'Le resultat,' says la Combe, 'a été des plus concluants.'

'Nevertheless,' it is added, 'the author does not admit that beri-beri is an alimentary disease due to a class of food of which rice forms the staple. He bases his opinion on what he observed in a Norwegian sailing ship, which landed a case of beri-beri on New Caledonia, and where rice formed no part of the food on the voyage. Already for several years cases of beri-beri had been noted on this vessel.'

Such a judgment is at once to affirm that all conditions of neuritis resembling beri-beri must be identical with it, and to deny that the disease (or others resembling it closely) can have more than one source. The beri-beri of ships, it will be shown later, comprises often a very distinct malady.

² *Gen. Tijdschr. v. Ned.-Ind.*, Deel xx. (N.S. ix.), Nos. 5, 6, p. 272, 1880.

DEPENDS ON QUANTITY OF RICE EATEN 241

Van Leent points out that the quantity of proteid shown by analysis (129 grammes) exceeds what was really available, the larger portion of it in the rice being with difficulty assimilable from that material. The flesh, he says, nearly always lean, yielded at all times but little fat, and often none at all. The dietary of the European crews at the same time was in every way better, and contained only 29 per cent. of rice, the absolute amount of this being 500 grammes. Their ration was as follows :

	Grammes.	Proteid.	Fat.	Carbohydrate.
Bread	400	30	—	184
Rice	500	38	—	400
Flesh	400	72	25	—
Lard	75	—	75	—
Butter	35	—	35	—
Beans or grey peas and potatoes }	300	40	—	150
Total	1,710	180	135	734

In addition, daily, greens, dried onions, coffee, tea, sugar, salt, pepper, vinegar, and grain.

During the years 1870, 1871, and 1872, while the ships lay off Banka and Borneo, beri-beri was very rife among the natives, while among the Europeans but few cases occurred. In 1874, when the vessels were at Atjeh, nearly two-thirds of the natives became attacked, and there was a trifling increase also among Europeans. The exact figures were :

Year.	Nationality.	Strength.	Cases of Beri-beri.	Proportion of Beri-beri per 1,000 of Strength.
1870	Europeans	2,259	14	4'7
	Natives	967	194	206'2
1871	Europeans	2,483	6	2'4
	Natives	831	206	247'0
1872	Europeans	2,326	19	8'1
	Natives	770	199	260'0
1873	Europeans	2,744	24	8'8
	Natives	762	460	603'7

Since no other measures served to check this appalling rate of sickness among the natives, 'as a last despairing regulation,' says Van Leent, it was ordered that native sailors should be no

242 THE CAUSE AND PREVENTION OF BERI-BERI

longer employed in Atjeh waters. They were replaced by Europeans, one for every two natives. This radical and, of course, complete remedy was, however, disapproved by the Dutch Home Government, who feared the danger of thus overworking their own sailors, and, on their urgent order, Javanese sailors were once more sent to Atjeh.

‘Meanwhile, the native *stokers* on the latter ships had been drawing European crews’ rations—a regulation which cannot be too much commended—and these were also extended to the Javanese sailors as soon as they rejoined their ships. . . . Never had any sanitary measure a success so complete as this. *Immediately the number of beri-beri cases fell very greatly.*’

In 1874 the official returns showed :

Nationality.	Strength.	Cases of Beri-beri.	Proportion of Beri-beri per 1,000 of Strength.
Europeans	2,810	2	0·7
Natives	722	51	70·6

‘These figures speak. Not to be convinced by them, people must have no desire to be persuaded. *And we can give assurance that beyond the radical alteration in the food of the native crews all other circumstances remained identically the same as before,*’ says Van Leent emphatically.

The next year (1875) afforded further proof of the beneficial effect of European diet on the natives.

There were extremely few cases of beri-beri on the ships *at Atjeh*. These were mostly relapses, or weakly individuals temporarily taken into service. But several ships were detached from the Atjeh Squadron for work elsewhere—to New Guinea and the Celebes—and on them the improved diet (still allowed on the Atjeh ships) was discontinued, the old meagre rations being reverted to. The result was that beri-beri increased again on these ships.

Year.	Nationality.	Strength.	Cases of Beri-beri.	Proportion of Beri-beri per 1,000 of Strength.
1875	Europeans	2,934	9	3·6
	Natives	903	129	142·8
1876	Europeans	2,786	1	0·4
	Natives	983	165	168·0

Again, little or no beri-beri *at Atjeh*. 'The issue of European crews' sea-rations daily to the natives had rooted out the sickness as by magic.' Some exceptions occurred, cases continuing to happen on one or two ships. A careful official inspection showed Van Leent that these were individuals who had not drawn the optional European ration, but preferred native diet. 'Therefore, European diet was made compulsory. From that moment beri-beri vanished, or almost vanished, on board these ships.'

'Meanwhile, fresh regulations for native crews were undertaken. Their circumstances generally were improved, and especially their food. This was brought on to a par in nutritive value with that of Europeans. . . . The new dietary came into force on January 1, 1878, and its wholesome results were not long in appearing.

'It is to be noted that it was at the end of 1877 that the natives came on to European diet. The following figures show the favourable effect of this wise regulation :

Year.	Nationality.	Strength.	Cases of Beri-beri.	Proportion of Beri-beri per 1,000 of Strength.
1877	Europeans Natives	2,500 1,100	7 123	2·8 111·9

'The last reports as to the good influence exercised by the introduction of the new regulation as to native dietary are very satisfactory. *Beri-beri has almost entirely disappeared* from our warships in India.'—1880.

The new diet for natives brought into force in 1878 was as follows (*loc. cit.*, p. 46) :

	Grammes.	Proteld.	Fat.	Carbohydrate.
Rice	1,000	75	—	800
Bread	200	15	—	92
Meat	400	72	25	—
Dried fish	100	18	10	—
Butter and cocoanut oil ..	45	—	45	—
Total	1,545	180	80	892

244 THE CAUSE AND PREVENTION OF BERI-BERI

With the adjuncts daily of greens, onions, coffee, sugar, tea, vinegar, pepper, salt. The rice in this diet forms 64.5 per cent. of the whole.

It seems impossible to refuse to this evidence the validity which Van Leent claims for it as, at least, proof of the dependence of beri-beri in some way upon diet.

The reduction of the disease among the natives on all ships at Atjeh when they were placed on European diet, its return in severity in those ships upon which the old scale of diet was reverted to, while the Atjeh Squadron still continued to enjoy almost complete immunity, are facts which have the force of an arranged demonstration.

The far greater incidence of the disease at all times on the natives, as compared with Europeans, is a conspicuous example of the direct proportion of the severity of the disease to the absolute quantity of rice eaten by the two classes. The reduction of cases among the natives when their ration of rice (remaining the same absolutely) was supplemented—diluted, neutralized—by more, and more generous food, illustrates remarkably the effect of relative decrease in the quantity eaten. On all this Van Leent rightly insists. But to the further conclusion which he draws from the facts—that they demonstrate the disease to be merely a dyscrasia, such as scorbutus—he defines beri-beri as ‘a disease of the blood, caused through bad feeding, owing to lack of sufficient quantity of proteid and fat in the diet’—must be opposed the considerations already urged against a similar interpretation of the Japanese, Chantabun, and other experiences.

As Van Dieren has pointed out, if beri-beri (in this as in other cases) depended merely on lack of fat and proteid, the disease should have been not merely reduced or diminished, but entirely eradicated, when the normal requirement of these elements was supplied, as Van Leent admits they were, both in the European diet at one time supplied to the natives, and in the new regulation ration diet of 1878. But, so far from the cases altogether ceasing, even during the best years, at least 20 to 30 per 1,000 of the native strength continued to be annually affected.

The conclusion is unavoidable that even with the best arranged diet physiologically the consumption of a kilo of rice daily will, if stale and uncured, produce poisoning in some individuals irrespective of all other conditions. Far better was the result

in the Japanese navy, where, when dietetic changes were adopted, they were made thorough. Not only was the absolute quantity of rice in the ration (never so great as in the Dutch Navy) made relatively less by the addition of fat, proteid, etc.—from forming 56 per cent. of the whole allowance it was reduced to 34 per cent.—but it was also absolutely lessened by one-sixth—from 782 grammes to 648 grammes—and the result was ‘radical.’

The same objection appears in, and is sustained by, the fact that, year after year, so many cases of beri-beri appeared among the Europeans, whose diet, as given by Van Leent, can certainly not be impugned on any physiological basis.

Worthy of note in this same connection is the contrast offered by the case of these Europeans in Dutch naval service with that of European crews serving under other flags. Van Leent's figures show that in successive years 4.7, 2.4, 8.1, 8.8 per cent. of the Dutch sailors suffered from beri-beri. Yet among British ships and sailors, a far larger number of whom constantly serve in the tropical zone, beri-beri is, if not entirely unknown, of the greatest rarity. The cause of this greater liability of the Dutch sailors is—can it be doubted?—the presence in their diet of 500 grammes of rice. Whence it may be inferred that even so little as 30 per cent. of this material, when of the sort supplied to the Dutch Fleet, will produce beri-beri, even where an ample supply of fat and flesh is also given.

The only other instances of beri-beri attacking *European* sailors on warships of which I have a note are:

(1) On the Italian royal naval vessel *Volturmo*, in which cases are described by Tiberio as occurring while lying off Zanzibar, March, 1901. There was not at the time, and had not for many years been, Tiberio says, any beri-beri among the natives of the district. Now, in the dietary which he gives of the crew it appears that *rice* was a regular component on four days out of seven.

(2) This author gives reference to three other epidemics on Italian warships related by Cavalli, Melardi, and by Moliterni. The rations on all Italian warships are alike, and thus rice was at fault in each of these cases.

(3) On H.B.M. ship the *Forte*, in 1900,¹ cases of beri-beri occurred among native ‘kroomen,’ but none of the white sailors suffered. The natives were fed on bad rice bought at Loanda.

¹ ‘Ann. Statist. Rep. of the Health of the Navy, 1900.’

246 THE CAUSE AND PREVENTION OF BERI-BERI

(4) On H.B.M. *Sphinx* in the same year,¹ in the Persian Gulf, there was an outbreak of beri-beri among white sailors. The medical officer attributed it to infected cabins, but it appears that rice was being eaten.

Van Dieren supplies the following further data as to the prevalence of beri-beri in the Dutch Navy after 1877 :

Year.	Nationality.	Strength.	Cases of Beri-beri.
1878	Europeans	<i>circa</i> 3,000	nil
	Natives	1,000	54
1879	Europeans	3,000	nil
	Natives	1,000	23

In 1880, he says, 'on twenty-one ships on which the native crews had European diet, with an average strength of 1,222 men, there were 39 cases of beri-beri ; on nine ships on native diet, and with an average strength of 307 men, there were altogether 56 cases.'

Other points there are worthy of note in connection with the Dutch Fleet epidemics. One of these is the contrast afforded in 1878, 1879, when improved diet had had effect in the Dutch vessels, between their condition and that of the Japanese Navy, in which similar measures, afterwards to produce such great results, had not yet been taken. More than one-third of the Japanese sailors were affected in those years. Another contrast is the very different state of affairs prevailing in the army operating in the same region (Atjeh) and at the same time.

13. **Beri-beri in Dutch E.I. Army.**—During the years when the epidemic in the fleet was at its worst (1870-1873), and again 1875-1877, not more than 8 per cent. at most even of the native troops of the army were affected. But in later years, when beri-beri had become so greatly reduced in the navy, the cases in the army steadily increased.

The importance of these facts lies in the opposition they afford to any suggestion that the fluctuations in beri-beri incidence in the fleet or army may have been due not to the dietetic changes, but rather to quite independent local or climatic or epidemiological factors, such as might determine, retard, or accelerate the march of maladies of the infective order.

¹ 'Ann. Statist. Rep. of the Health of the Navy, 1900.'

It is remarkable that with the convincing object-lesson of the results achieved in the Dutch *Fleet* at this time there should have been made, or that, if there were made, there should have been recorded, no application of similar measures to prevent the great losses which were suffered by the Dutch *Army* almost contemporaneously, and in the same region, from the disease.

In spite of the importance of the subject, I fail to find in the principal writings of Van Leent, Van der Burg, or Van Dieren, or in Laoh's recent brochure, any account either of the diets of the different races of soldiers or of the changes which, it seems certain, must have taken place to account for the enormous fluctuations in the case incidence of beri-beri among them.

Statistics compiled by Van der Burg,¹ and some comments by Van Dieren,² supply the following data indicating that a great effect was produced by alteration in the diets of the Dutch Army. From 1873, when the war against Atjeh began, to 1884, the strength of the Dutch troops engaged there and the incidence of beri-beri upon them had been as follows :

Year.	Strength.		Case-incidence of Beri-beri per 1,000.	
	Europeans.	Natives.	Europeans.	Natives.
1873	18,000	14,200	5	49'1
1874	11,000	15,500	6	56'4
1875	15,600	16,500	4'1	82'4
1876	15,000	19,200	3'1	52'8
1877	15,500	22,900	4'3	76'1
1878	17,500	19,300	2'2	122'8
1879	15,000	16,000	4'4	275'0
1880	16,000	15,000	4'2	267'7
1881	15,500	14,500	3'7	399'1
1882	15,300	14,500	4'3	208'6
1883	14,400	14,400	15'4	251'8
1884	14,900	15,900	10'1	326'2

'On July 1, 1885,' says Van Dieren, 'a ration almost identically the same as the "shocking" ³ navy ration, the so-called "famine-allowance," was instituted in the army. The consequences were appalling. In the summer reports we read [but in them nothing is said about the change of food]: "During the last four months especially of 1885 beri-beri claimed many

¹ 'Weekblad,' *v.h. Ned. Ind. Tijdsch. v. Gen.* 3, January 1, 1896, p. 88.

² 'Beri-beri een Rijstvergiftiging,' etc., p. 93.

³ 'Angstaanjagende.'

248 THE CAUSE AND PREVENTION OF BERI-BERI

victims among Europeans." "The sickness-rate, which in 1884 was no more than 1.03 per cent., rose to 7.07 per cent., and in 1886 to 25.9 per cent."

'At the end of 1886 there was "disinfection," and the sickness-rates were :

1887	13.4 per cent.
1888	11.7 " "

'Professor Pekelharing concludes "that it was the disinfection that had driven off the sickness, since no one had discovered any other reason for the improvement."

'In the *Dagblad v. Zuid-Hollanden en 's Gravenhage* the retort was made by me to this that *besides* the disinfection on October 21, 1886, a new dietary was brought into force by General Demmenie in which more proteid and fat were contained.'

The corrected figures of Van der Burg, published in 1896, show:

Year.	Strength.		Case-incidence of Beri-beri per 1,000.	
	Europeans.	Natives.	Europeans.	Natives.
1885	14,800	14,300	122	617.2
1886	14,300	13,600	258	445.2
1887	15,300	15,800	134	303.3
1888	16,600	17,200	117	360.0
1889	16,200	17,500	79	265.4
1890	14,700	17,000	91.8	273.4
1891	14,500	18,300	74	230.0
1892	14,800	18,600	86.5	237.5
1893	15,100	19,000	84.5	271.6
1894	11,600	20,800	71.4	204.0

With an average force of less than 15,000 Europeans the Dutch had in these twenty-two years (1873-1894) 17,520 cases of beri-beri, of whom 356 died. Among the natives (average force under 19,000) there were 85,881 cases, of whom 5,308 died.

Taken alone, the results noted by Van Dieren might be held to be, as might every other such change accompanying alteration of diet which has ever been recorded, a mere coincidence, really the effect of a cause operating independently of food and all local factors. But the repetition of the coincidences, and especially the reversal of the effect, when the mischievous depravation of diet was again undone, leaves it little doubtful that they were causally connected with—in fact, directly due to—

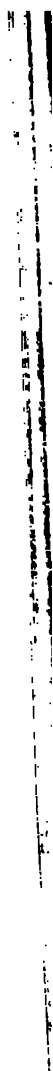
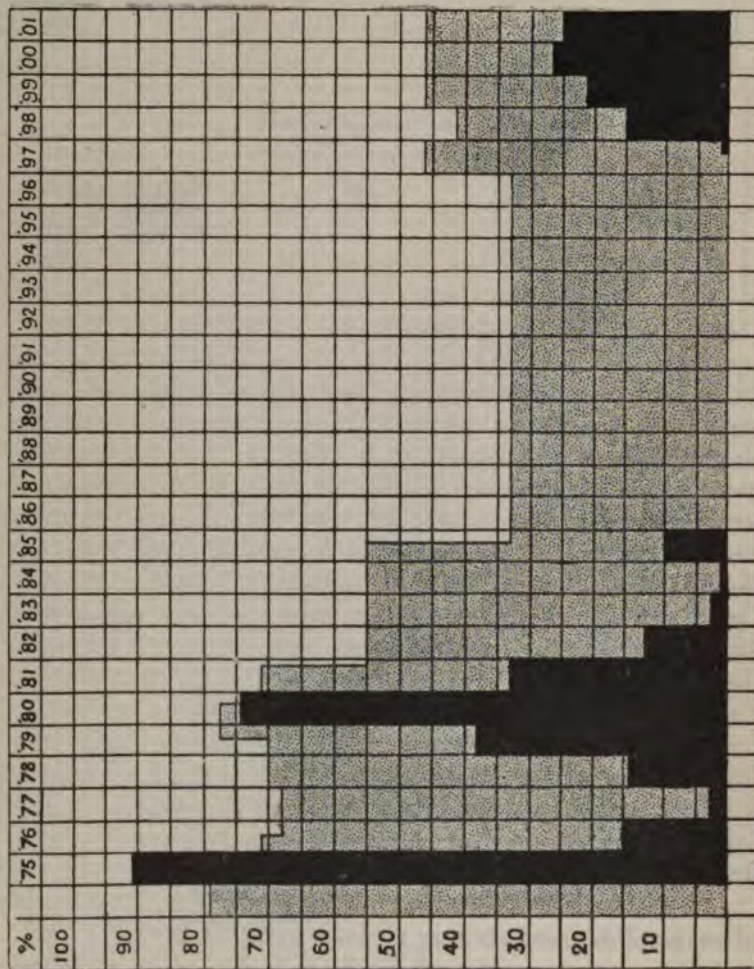


CHART SHOWING CASE-INCIDENCE OF BERI-BERI, AS DEPENDENT
ON PROPORTION OF RICE IN DIET, IN SINGAPORE GAOL, FROM
1875 TO 1901.



The portion lightly shaded represents the proportion of rice in daily diet of prisoners ;
the darker shading the case-incidence of beri-beri, or proportion of cases of beri-
beri per 1,000 of daily average strength.

To face page 251.

DEPENDS ON QUANTITY OF RICE EATEN 251

total diet, it was reduced to 14 ounces, forming only 33 per cent. of the whole daily ration.

When this low proportion was reached, the disease disappeared ; and *so long as the same ratio was adhered to the gaol remained free from beri-beri.*

When, in 1897, the absolute quantity, and relative proportion also, of the rice in the diet was increased (to 19 ounces, forming 46 per cent. of the whole ration), beri-beri became evident again.

Many changes were made in the prison dietaries from time to time (full analysis of which will be found in the Appendix), the result of which was that, in each case, when the proportion of rice was increased, so was the prevalence of beri-beri, and conversely, when the rice was reduced, the disease diminished also.

The following figures show the proportion of rice in the whole ration, and the case-incidence of beri-beri per 1,000 of strength year by year from 1875-1899 :

	Rice in Daily Ration.		Beri-beri.
	Actual Quantity in Ounces.	Proportion per Cent. of Whole Ration.	Cases per 1,000 of Strength.
1875	28	79	921
1876 (to July)	28	71 }	159
1876 (July to December) ..	28	68 }	
1877	28	68	
1878	26	70	very slight
1879 (January to November)	26	70 }	153
1879 (November to December)	26	77 }	388
1880 (January to July) ..	26	77 }	748
1880 (July to December) ..	24	71 }	
1881 (January to September)	24	71 }	
1881 (September to December)	24	55 }	332
1882	24	55	126
1883	24	55	28
1884	24	55	10
1885 (January to July) ..	24	55	98
1885 (July to December) ..	14	33	nil
1886 to 1897	14	33	nil
1897 (September)	19'4	46	5
1898	19'4	46	154
1899	19'4	46	213
1900	19'4	46	266
1901	19'4	46	252

The fluctuations in both factors appear more clearly in the attached chart, in which the alterations in the quantity of rice in

252 THE CAUSE AND PREVENTION OF BERI-BERI

the diet (percentage of whole ration) and the proportion of cases of beri-beri occurring per 100 of daily average strength are shown.

It is clearly seen that only when the proportion of rice exceeded 33 per cent. did beri-beri appear. It is remarkable that it was only when the same proportion was reached also that the disease ceased to appear in the Japanese forces.

15. Selangor Prison Epidemics.—In the Selangor prisons (Old Gaol, Kuala Lumpur, and New Gaol, Pudo) beri-beri also prevailed from the earliest days. From 1883 to 1892 the daily average population was under 200, but the following cases occurred :

	1883.	1884.	1885.	1886.	1887.	1888.	1889.	1890.	1891.
Cases of beri-beri	36	12	3	—	11	12	26	14	9

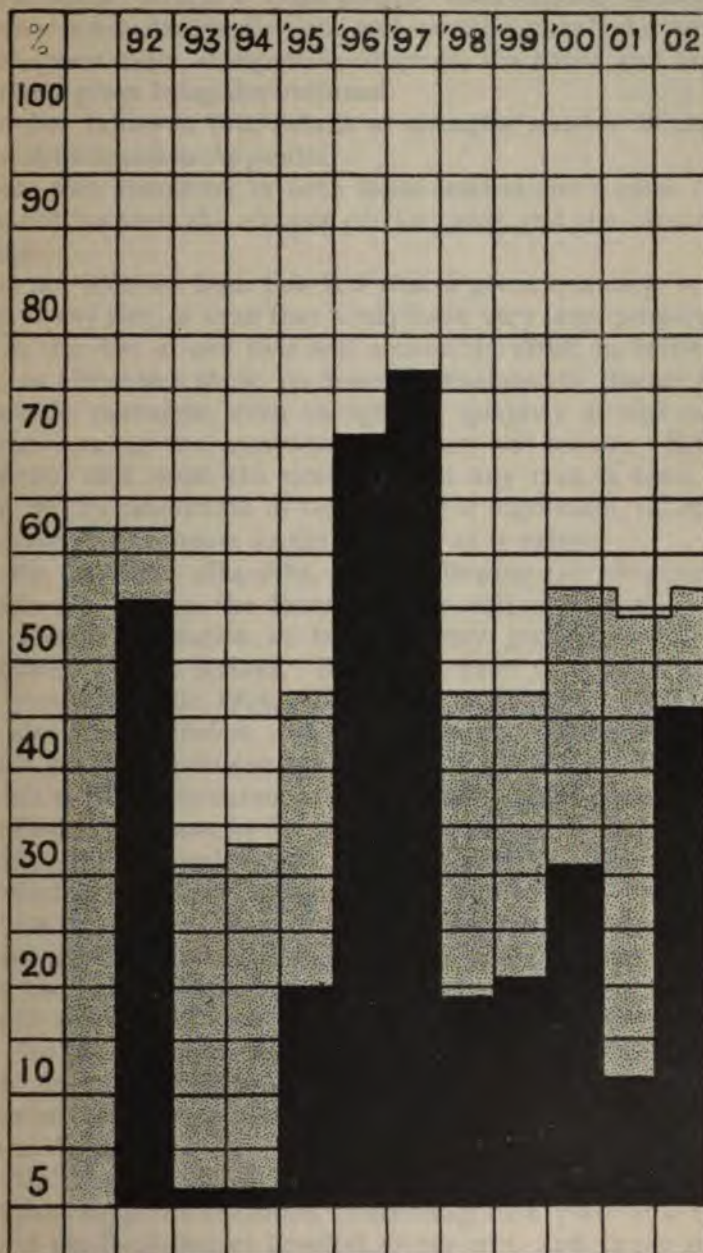
The Resident, referring, in 1884, to the 'constant presence of beri-beri in the gaols of the State,' reported that a new diet had been adopted which had at that date (early in 1884) produced no change. The effect of this alteration seems to have been felt in 1885 and 1886.

I have no exact records of the earlier years, but from 1892 the effect of various alterations in the quantity and proportion of rice in the daily ration and the incidence of beri-beri appear as follows :

Year.	Rice in Daily Ration.		Beri-beri.
	Absolute Quantity in Ounces.	Proportion per Cent. of whole Diet.	
1892	21'3	62	56
1893	16'57	31	14 ¹
1894	14'00	33	14 ¹
1895	19'14	47	200
1896	19'14	47	709
1897	19'14	47	767
1898	19'14	47	189
1899	19'14	47	207
1900	22	57	315
1901 (January to April) ..	22	57	119
1901 (May, to April, 1902) ..	21	54	
1902 (May to December) ..	22	57	

¹ It has been stated by Travers that the few cases occurring in these years did not originate in the gaol, but were imported—a fact which makes the reduction of rice from 62 to 31 per cent. appear of the greater effect.

CHART SHOWING CASE-INCIDENCE OF BERI-BERI,
AS DEPENDENT ON PROPORTION OF RICE IN
DIET IN SELANGOR PRISON FROM 1892 TO 1902.



The portion lightly shaded represents the proportion of rice in daily diet of prisoners; the darker shading the case-incidence of beri-beri, or proportion of cases of beri-beri per 1,000 of daily average strength.

To face page 252.



The period from May 3, 1901, to April 30, 1902, was that of Dr. Hamilton Wright's experimental observations, when no salt fish was admitted to the gaol, and every prisoner had 6 ounces of fresh meat daily, irrespective of grade, the beans and bread previously given being discontinued.

The diet tables in full, details of strength, number of cases, etc., will be found in Appendix.

There was, therefore, in both these institutions a clear concomitance between the amount of rice eaten and the extent of beri-beri.

It is not inferred from this fact that a given quantity, or an excess of *any* rice, or even that a relatively very large proportion of it in the diet *at any time* will necessarily result in beri-beri, since, as the tables show, on several occasions the disease was not greatly prevalent, even though the quantity of rice eaten was the same as in years when beri-beri was severe. But it is asserted that when the rice in use at any time is toxic (as shown by the production of beri-beri by it anywhere), its effect is directly proportionate to the quantity of it eaten.

At the juncture, 1884-1885, when, following the adoption of an improved dietary, the disease became reduced in both gaols, there was a diminution in beri-beri very generally observed throughout British Malaya. Similarly, when it became again most prevalent (after 1895), this was so everywhere outside the gaols also. Nevertheless, the *number of occasions* upon which no such general conditions concurred to explain the result, and yet a fall or rise in the extent of beri-beri in the gaols accompanied a decrease, or increase, in the quantity of rice eaten, is sufficient to justify the conclusion that the changes in the diet actively influenced the incidence of the disease.

In the case of the Singapore prison, for twelve years entire freedom from the disease was enjoyed (on a minimum amount of rice in the ration), while beri-beri, as shown by hospital records, was still a source of constant and extensive illness everywhere else. It thus continued all this time unabated in the neighbouring lunatic asylum. It formed one-fifth of all the admissions to the hospitals—thirty-eight in number—of which statistics are given in Appendix.

At the Kuala Lumpur Prison the first decline in beri-beri accompanying a rice-reduction (1893-1894) took place at a time when at the local pauper hospital 37 per cent. and 32 per cent. respectively of all the admissions were for beri-beri.

Thus, though strictly concomitant with alterations in the proportion of rice in the dietaries, beri-beri-incidence in the gaols failed to an important extent to correspond with fluctuations in the prevalence of the disease *outside*, and hence may be inferred to have depended on the only condition in which there was recognised alteration—namely, on the dietetic changes. In other words, *the cause of the disease being abroad*, the prison experiences show, *its incidence on prisoners varied directly with the quantity of rice eaten*.

16. The Singapore Lunatic Asylum.—If 20 ounces of dry rice, of such an uncured sort as is here held to be toxic, even when consumed with a pound of other adjuncts, such as flesh and vegetables, making a diet 'physiologically correct' (as is the case in the Kuala Lumpur and other prisons of Malaya), is sufficient to cause beri-beri, what is likely to be the effect of consuming 2 *pounds* daily?

The only institution in the States and Settlements at which, so far as I know, nearly so large an allowance of rice is given to the inmates at the present date is the lunatic asylum in Singapore, and the state of this institution,¹ which has been described by its superintendent as a 'pesthole of beri-beri,' furnishes an answer to the question.

The condition of the Dutch and Japanese navy, army, and prisons, of the prisoners in the Singapore gaol before 1884, of the Annamite and Japanese emigrants into New Caledonia, in all of which instances the allowance was about a kilo (2½ pounds) of rice daily, afford similar proofs of appalling effects following the consumption of such large quantities.

17. Japanese Coolies in Fiji.—The epidemic described by Noble Joynt among Japanese coolies imported into Fiji, of whom 226 out of 250 were attacked, 69 dying, affords an example of the effect of excess of rice, as well as of the dependence of beri-beri upon the *sort* of it eaten. The dietary of these men is stated to have been 'liberal,' including ½ pound of meat or fresh fish daily, but the allowance of rice was 2 *pounds*. Now, this is a quantity which the Japanese coolie in his own country seldom earns enough even to purchase, and certainly is not accustomed to consume.

In regard to this epidemic, the following significant facts, which have been already mentioned (p. 110), may be recalled:—

¹ Written in 1903. Since that date great amelioration has taken place as to the part played by diet, in which I have as yet no certain information.

The Japanese quarters were newly built specially for them to occupy on arrival, and it was a condition of their contract that for the first six months' residence all their rations should be imported from Japan. Later, however, they were given rice which was of the common stock issued to the Indian coolies and native labourers, none of whom ever acquired beri-beri. The Japs also used some bread, fresh meat, sugar, and salt, from local sources ; but, with those exceptions, and the rice after six months, all their diet was imported from Japan specially for them.

Patients in hospital were given ordinary local diets not imported from Japan.

The Japs did not work in the same field batches as Indian coolies or Fijians ; but after working hours there was absolutely no bar, except social differences, between the Japs and all other nationalities on the estates, or in the neighbouring villages, and it was an everyday occurrence for them to stroll about together.

The rice imported from Calcutta was ordinary Ballam rice such as is used in Fiji year after year.

No person in Fiji has had beri-beri except the Japs, and in 1904 a couple of newly-arrived Chinese merchants' employés.

This epidemic alone, therefore, afforded unerring evidence of the true source of beri-beri. Between the Japanese coolies who were selected and left their country healthy and free of disease and the several races inhabiting the country—beri-beri free—into which they were imported, and where they all associated freely, there was from first to last no other, or no specific, difference except that of food. But there was between them this solitary distinction. Those who got beri-beri were, as they always had been, fed upon the rice—stale and uncured—customary in their country. Those fed on Bengal (cured) rice escaped the malady, as they had always done.

18. Case of the Dutch Army and Fleet.—Allusion has been made to the statement that in the Dutch Army in Sumatra, both European and native, beri-beri was more prevalent among those in garrison than when on active service, and it has been pointed out that of this an explanation may be found in the fact that the diet of both classes contains 20 per cent. more rice when in garrison.

The statement of Overbeck de Meijer¹ has also been cited,

¹ Quoted from Hirsch, 'Hist. and Geog. Path.,' vol. ii.

'that whenever the troops employed on military expeditions into certain parts of the Dutch East Indies are obliged to live exclusively on the ordinary food of the country, they are almost always attacked by beri-beri.' The 'ordinary food of the country' in Sumatra is, of course, chiefly rice.¹

As regards the fleet, the testimony of Van Leent, already given, is very clear and decisive as to the effect of the relative amount of rice eaten in producing beri-beri.

19. **Special Incidence of Beri-beri on the Well-fed.**—In differences of quantity of rice eaten is to be found also the explanation of a feature, peculiar to beri-beri among diseases, which has been commented upon by most authorities, but of which no satisfactory explanation has ever been offered—the tendency, namely, of the disease to attack especially the otherwise healthy and well-fed, rather than the sickly: the young and robust, or even the fatter among their fellows, more than the lean and weak.

The consideration that the vigorous and strong naturally eat rice in quantities absolutely greater than do the sickly, affords, however, a ready reason for their more often acquiring the disease; and, what is of more importance, explains how, for the opposite reason, the liability of the weaklier individuals to the disorder should be diminished.

20. **Its usual less Incidence on Females.**—The well-known lower incidence, under ordinary conditions, of the disease upon native women is due probably to similar causes, since there is no doubt that the women, who feed everywhere, *more orientali*, after their lords, get both absolutely and relatively smaller amounts of food than they.

In prisons, asylums, etc., where the rations given to both sexes are equal, no difference in the incidence is to be observed. But I have noticed that *amahs* (who feed themselves, and can afford to buy plenty of rice), the Chinese wives of Eurasians, and Straits-born Chinese 'babas,' are peculiarly prone to the malady. This is true also of prostitutes. All these classes eat rice in excess.

21. **Quantities Normally Innocuous made Toxic under Changed Conditions.**—In the above instances the amount of rice eaten has been

¹ It must not be supposed, however, that the Dutch troops enjoyed fresh ('red') rice. This the natives of a hostile country would have been hardly willing to prepare for them, and it would have been too tedious for the troops themselves to do so. The rice which the troops ate was white shop (uncured) rice, which they carried with them.

generally large, and changes in the incidence of beri-beri have followed alterations in the absolute quantities of rice eaten.

But in a very large number of instances (it may be shown) the absolute quantity of rice eaten may remain the same, or even be diminished, and yet through the absence of other natural components of food in right proportion, have an effect relatively as great as, or even greater than, larger doses of the same rice would have had, taken in combination with the proper quantities of other dietetic elements.

It is well known that the effect of alcohol and many other poisons is thus masked or diminished by the simultaneous ingestion of other food. Hirsch quotes from Puchstein¹ an example of ergotism which seems apt in this connection. In one village (Stregow), when all were using the same rye, five persons, all in one shepherd's family, had the disease. The neighbours remained free. On investigation, it appeared that the latter had mixed with the flour containing ergot a quantity of potatoes and barley, whereas the shepherd's family had eaten the ergotized flour unmixed.

It would not be deduced from this that potatoes are an antidote to ergot, or that it is to lack of potatoes in diet that ergotism is due; yet it was through the absence of potatoes that the effect—'ergotism'—was produced in this particular instance.

It thus becomes conceivable how, in the case of poisonous rice, restitution to the diet of any elements, whether proteid or fatty, in which it has been deficient, may check the production of beri-beri, not so much because the disease depends on the want of them as because the relative amount of the rice consumed has been thereby made less. The additions to the diet in such cases serve, in fact, less as antidotes than as *diluents*.

22. The Effect of Fat—Laurent—the Chantabun Epidemy.—Laurent relates remarkable results from the addition of *fat* to a diet in which it has been lacking. An epidemy of beri-beri broke out among Annamite soldiers at Chantabun in Siam.²

Fifty-two cases suddenly appeared among 260 soldiers. Laurent, acting on the view (derived from Brémaud) that want

¹ *Loc. cit.*, 'Ergotism.'

² *Arch. de Méd. Nav.*, March, 1899.

258 THE CAUSE AND PREVENTION OF BERI-BERI

of fat in the dietary was the determining cause, took steps to have this important article added. The men, whose rations included twelve meals of fresh beef, and two of preserved beef weekly, had complained of the beef being too lean to eat with their rice. A new scale, including eleven rations of fat pork, with fresh vegetables, and three of fresh beef weekly, was issued, and the epidemy ceased 'in forty-eight hours'; those affected began to recover, and no new cases occurred.

No difference was made in the relative proportion of rice in the whole diet, except, perhaps, such as the addition of fresh vegetables entailed. The real alteration was the substitution of fat meat for lean.

The interesting facts appear in this epidemy that of the 260 native soldiers attacked—

140 were bachelors, and of them			
100 had only the service rations;			
and among them there were ..	47 cases, or 47 per cent.		
40 boarded out, and got extra			
articles of food; and among			
them there were	3	„	7'25 „
120 were married men, and they			
furnished	2	„	1'66 „

Among the 120 wives no cases happened.

Between the married men and the bachelors the only difference lay in the food. The quarters of both classes were similar, their work and exposure to chill equal. Water was the same for all. The married men were naturally the more crowded, and probably had each absolutely less food, since their rations were shared by wives and children. They had more oily food, since the women added fat, etc., in cooking.

Laurent, although entirely convinced that the addition of fat to the dietary of the soldiers stopped beri-beri on this occasion, recognises that absence of fat was not the sole cause of its production, since, he points out, the worst case was a Frenchman—the only European attacked—who got sufficient fat, and 'he rapidly recovered on removal.'

23. Brémaud on Poulo-Condore Epidemy in 1878.—Brémaud, commenting on Laurent's epidemy, relates his own experience of the value of fat in preventing beri-beri. He states that Poulo-Condore, a large convict settlement containing 1,000 or more prisoners, was, previously to 1877, a 'veritable sana-

torium.' As prophylactic measures against beri-beri, the convicts were allowed great freedom, keeping pigs, poultry, and gardens, and altogether were treated like colonists rather than convicts. Under a new Governor all this was changed. The prisoners were closely confined in cells, in complete inaction, and the rations became worse, 'consisting for the most part of water-cooked rice and a little salt.' Under these circumstances, a severe epidemic of beri-beri broke out, with many deaths. On the resumption of the original fat-containing diet the epidemic immediately ceased.

24. **Second Epidemy, Poulo-Condore, 1897-1898, ceased on adding Fat to Diet (Laurent).**—Following a second reversion to worse diet, the disease once more appeared at Poulo-Condore. Laurent gives details of the second epidemic in 1897-1898. The mortality from beri-beri, which was at the rate of 10 per 1,000 *per mensem* previously, rose rapidly, becoming in—

November, 1897 ..	32 per 1,000.
December, 1897 ..	58 per 1,000.
January, 1898 ..	130 per 1,000 (72 deaths among 767 prisoners).
February, 1898 ..	148 per 1,000 (67 deaths among 499 prisoners).
August, 1898 ..	24 per 1,000 (8 deaths among 335 prisoners).
September, 1898 ..	55 per 1,000 (18 deaths among 323 prisoners).
October, 1898 ..	25 deaths.
November, 1898 ..	30 deaths.

The disease continued to decrease after November, although 'still in the prison.'

Laurent, visiting the place in October, 1898, caused 250 grammes of fresh pork to be added daily to the rations, except two days a week, when 350 grammes fresh fish were substituted. Vegetables and condiments continued as usual.

'Following on this, the number of cases of beri-beri declined,' though, as it would seem, not immediately.

It may possibly be of some significance in connection with the apparent efficacy of fat in relieving or preventing beri-beri that *oils* are reputed useful as antidotes for many poisons of an irritant class. The writer has given prominence to the instances advanced by the French observers, in proof of the influence of *fat* upon the production of beri-beri, upon account of the ardour of the convictions expressed by them as to the reality of that effect. But it will be observed that both at Chantabun and Poulo-Condore, when the amelioration of the diet took place,

by which beri-beri was controlled, not fat alone was added to the rations. In each case other articles were added, and so, if not the absolute, the relative, amount of rice eaten was reduced. This change alone might very well have accounted for the event, both in these instances and those which follow.

Extensive observations upon patients, to whose diet cod-liver oil, butter, or fat pork was specially added, have convinced the writer that, in persons already affected at least, the disease is little, if at all, influenced by the greater or less proportion of fat in the diet.

25. **Epidemics among Africans in Madagascar.**—Petit,¹ describing epidemics among African troops employed in Madagascar in 1898-1899, remarks that the cases occurred when oil, fresh meat (500 grammes daily), fresh pork, and other adjuncts were taken in plenty, when, also, no salt fish was used, so that the disease could not be due to either fat or nitrogen starvation, or to fish. He traces it to bad rice, and cites experiments of his own to prove that when *padi* (undecorticated and therefore *fresh* rice) was added in a certain proportion to the ration no beri-beri occurred.

For 100 grammes of the stale rice in the allowance 300 grammes of *padi* were substituted and added (after some opposition) to the rest of the rice. It was partly peeled (*demi-pilé*) first, then mixed, and boiled with the white rice.

Petit offers these facts as affording confirmation of a view of Eijkman (discussed elsewhere) that the poison in rice is neutralized by an antidote in the husk.

But in Petit's experiment the ration was *reduced* by the 100 grammes of stale rice taken off, was thus absolutely less, and further *diluted* by addition of 300 grammes of fresh rice. This reduction and dilution it was, in the writer's view, which determined the beneficial result.

26. **Epidemics in French Guinea arrested by Improved Diet.**—The following instances, related by Pinard and Boyé² in a review of the medical geography of French Guinea (tropical East Africa, latitude 7° 40' to 12° 40', longitude 10° 20' to 17° 30' W.), illustrate further the influence of relative proportion of rice eaten in the production of beri-beri :

'BERI-BERI.—At the end of 1900 a considerable number of

¹ *Ann. d'Hyg. et Méd. Col.*, 1903, vi., 1, p. 98.

² 'Guinée Française,' par Dr. E. Pinard et M. le Dr. Boyé, *Ann. d'Hyg. et de Méd. Col.*, t. vii., No. 4, October-December, 1904, p. 516.

cases of beri-beri, resulting in three deaths, one *foudroyant*, appeared among the Senegalese Rifles stationed at Conakry. The diet of these soldiers is excellent, but after inquiry it was proved that they sold the meat allotted them in order to gratify their gambling propensities. Strict measures were taken to stop this traffic, and from the day upon which they ate their meat ration, the disease completely vanished.

'In 1902 other cases, similarly followed by some deaths, happened among the labourers on the railway, treated most of them at the railway ambulance, except a few who were sent to Conakry. They quickly got well, thanks to change of diet, with diuretics and aperients. The diet was increased. Beef was distributed three or four times a week. This dieting brought the epidemic to an abrupt termination.

'We do not wish,' say these writers, 'to maintain that beri-beri is simply alimentary in origin, but we made a point of noting (*nous tenions à signaler*) the coincidence of sudden cessation of these pseudo-epidemics with alteration of diet. These facts prove,' they go on somewhat inconsequently to add, 'that beri-beri is not due to any poisoning caused by rice, since this continued to be issued with the additional meat rations!'

27. **Cases prevented by Improved Dietary.**—At the Tan Teck Seng (or pauper) Hospital in Singapore the numerous attacks occurring among patients admitted for other ailments ceased to appear, Von Tunzelman says, after the addition of a liberal allowance of flour to the dietary¹.

28. **Epidemy on a British Ship stopped by Fat.**—Archer, a British naval medical officer (who was one of a committee appointed by the Straits Settlements Government to report on the serious outbreak of beri-beri then prevailing in the Singapore Prison)² stated that an epidemy on board a ship in his care owed its occurrence to conditions, and was brought to an end by measures, similar to those Pinard and Boyé described as prevalent at Conakry. In Archer's case the sailors had a cash allowance given them for the purpose of buying fat and other necessary adjuncts to their rations. This they saved, instead of using. When the cash was stopped and actual commodities were supplied, no more cases of beri-beri happened.

In one of the epidemics described by Schuttelaere at Diego Suarez, mentioned earlier, it will be remembered he

¹ *Lancet*, December 22, 1894.

² See Straits Settlements Blue-Book, 1880, p. 97.

states that an issue of fat added to the rations ended the outbreak.

29. **Where Prison Officers suffered more than Prisoners.**—Miura¹ long ago made the observation—a singular exception to the usual order of events in such institutions—that in ‘the Japanese penitentiaries, which use a barley diet freely, cases occur with great rarity among the prisoners, while many officers and physicians had it. Many prisoners arriving with it quickly recover from it there. This Miura attributes to the barley diet,’ and he goes on to show, says Bentley,² from whom I quote this, that the factor common to both the prisons and the Navy (then on the reformed diet) which prevented the entry of the Kakké poison—which Miura believed lay in certain sorts of fish—was ‘the exclusion of regular fish food from the diet.’

For us, of course, the fact forms one more example of the dependence of the disease upon the *quantity* of rice eaten. For the officers and physicians here had plenty of rice, but the prisoners, fed on cheaper *barley*, had less.

30. **The Japanese Mercantile Marine.**—Saneyoshi relates that during the Japan-China War many merchant vessels were chartered, on each of which a few sailors from the navy were added to the ordinary ship’s crew. ‘On each ship the naval men had exactly the same duties as others, but the former were served with the navy regulation diet, while the original crews of the ship had their own. The seamen of the navy were entirely free [from beri-beri], while many of the sailors belonging to the ships were attacked.’

On the Nippon Yusen Kaisha line, he states, beri-beri was frequent until improvement in the sailors’ diet was effected, when such cases ceased.

In the former case the observation has the value of a control experiment, since the men who got beri-beri could be compared with others circumstanced exactly like themselves, except that the proportion of rice in their diets was less.

31. **Debtors and Others in Lock-up in Batavia.**—Vorderman has related the following effective case :

‘At Batavia the block in which *gegijzelden voor schuld* (or persons incarcerated for debt) were kept, and where some of them were detained for as much as three years, was the same as that in which natives were confined who were held in preliminary detention by the *schout* (or police officer), Van Glodok. . . .

¹ *Virch. Arch.*, Bd. cxiv., *Heft* 3.

² ‘Beri-beri,’ p. 28.

Among those so detained—the *schouts-arrestanten*—beri-beri appeared, while the debtors, although they had been, on the average, far longer in confinement, remained free of the disease.

‘Both classes of persons lodged beneath one roof, and were separated from each other simply by a party wall.

‘The *gegijzelden* ate “stripped” (uncured stale white rice, exactly the same as the *schouts-arrestanten*, but they were able to send for food to their own homes, and to add a variety of extras which were lacking from the rations of scoured rice (*zonder zilvervries*), thus improving their powers of resistance; while the *schouts-arrestanten* could not do this, but had to be content with the ordinary prison fare.’

32. **Debtors at Waterplaats Prison.**—Van Dieren relates¹ that at the Waterplaats Prison in Batavia, where, during 1857 and the following years, half or more of the strength of the native convicts suffered from beri-beri, the *Chinese debtors*, confined in the same building, and the Europeans were free from the disease.

The debtors here, also, supplied themselves, while the Europeans had different fare.

In this gaol the sickness-rate from all causes was in 1857 so great that in March of a total strength of 346 persons, 327 were sick. This terrible condition had been attributed by the medical officer, Swaving,² chiefly to overcrowding, which steps were taken to reduce. But two days before the reduction of the strength, an additional ration of 1 pound of flesh daily was given to the inmates. Thereafter disease declined until, from a proportion of over 90 per cent. sick (mostly with beri-beri), the rate was reduced to an average, for the nineteen months following, of about 25 per cent. In October of 1858 the extra meat-ration was reduced by a half, and at once the sick-rate began to increase again rapidly, so that throughout 1859 it averaged 60 per cent., and sometimes reached over 90 per cent. of the strength, as before. And this happened although the number of occupants was then but half the original strength.

33. **The Prisoners at the Magelang Military Hospital.**—This episode, which has already been referred to in Part II. of this section, afforded, in Vorderman’s view, such strong evidence against any rice-toxin theory of beri-beri, that in a *naschrift*

¹ ‘*B.B. eene Rijstvergiftiging*,’ p. 79.

² Swaving, ‘*De oorzaken en de gevolgen der ongezondheid van eenige gevangenissen en hospitalen op Java.*’

to his work he puts it forward in detail as an answer to Van Dieren's views, then recently published.

It will be remembered that the occurrence was that a certain number of prisoners working at the military hospital, who, so long as they had been fed on red rice had remained free of beri-beri, acquired it when that rice was changed for white.

'This rice was of one and the same quality as the servants of the hospital establishment used, and was cooked in the identical pots in which the rice destined for the staff was cooked.

'Moreover, the same white rice served for the diet of the local garrison.

'Now, with this rice for their food, beri-beri appears in 10 per cent. of the convicts, in very few of the soldiers in garrison or hospital. Yet, according to Dr. Van Dieren's theory, this rice must have had poisonous properties.

'In 1895 there were 5,262 sick admitted into the military hospital at Magelang. Among the patients were 3 Europeans and 5 natives with recurrent beri-beri (*recidivisten van beri-beri*), of whom 2 Europeans and 1 native had been transferred from elsewhere.

'In addition, 1 native acquired primary beri-beri in garrison, while it attacked 2 other soldiers who were admitted to hospital for other maladies while there.

'The strength of the garrison in 1895¹ totalled 1,770 Europeans, 3 Africans, and 1,325 natives, or 3,098 men, among whom thus—not reckoning the 3 transfers—5 relapses and 3 primary cases of beri-beri happened—*i.e.*, 0.161 per cent. of the garrison with relapses, and 0.097 per cent. with first attack.

'Bear in mind that the so-called toxic rice, which the people used who fell sick, also served for the food of all the others.'

Vorderman goes on to say that while such an episode as the poisoning of some individuals only among many who had eaten together what were apparently all the same oysters might be easily explained—in the fact that some only of the oysters themselves were bad—such an explanation could hardly be invoked in the case of separate rice-seeds, '... so that the extremely scarce beri-beri cases, as they happened in the garrison and hospital of Magelang, are hardly explicable through any theory of rice-poison.'

'It must be admitted, too, that the rations (*hooftvoeding*) of

¹ By what is doubtless a misprint in his text Vorderman refers to this year as 1885.

the soldiery at Magelang at that time were such that (*with very few exceptions*) they were enabled, with the help of proper adjuncts, (*middels compensatieve neenvoeding*), to prevent the onset of beri-beri.

'How the case may have been with the diets of those particular individuals who were attacked by beri-beri cannot, however, very well be made out, since it is never perfectly possible to say that a soldier uses up all the articles of food given him.'¹

It has seemed worth while to detail the circumstances of this small epidemic at some length, since it is not only an instance perfectly to the point in this section of this work, but it is a type of many similar instances upon which objections just such as Vorderman urges have been laid by various observers against rice as a medium through which beri-beri may be incurred.

For the reason why the prisoners in this institution got beri-beri from their rice, although all the rest of the community among whom they worked, and who ate the same rice did not do so, is evident. The prisoners got the disease because they ate *more* rice, if not absolutely, yet in proportion to the rest of their diet than did the soldiers.

Their fare was prison fare,² in which rice ($\frac{3}{4}$ kilo daily) bulked to the amount of from 61 to 72 per cent. The rations of the soldiery may have contained a kilo of the same rice, but it was supplemented so largely with bread, flesh, fat, and other extras that the evil effects of the rice were largely reduced—whether by mere dilution, or through the antagonistic effect of proteids or fat against the beri-beri.³

¹ 'Onderzoek,' pp. 135, 136.

² The diet in the Java prisons was as follows :

			Kilogrammes.
Rice	0'75
Salt	0'02
Spanish pepper	0'04 (one)
Flesh	0'25
Or dried fish	0'12
Or dried meat	0'12
Vegetables	0'15
Total...	1'23 (containing 61 per cent. rice)
Or...	1'03 " 72 " "

(The diets of the navy are given on p. 243.)

³ I have no note of the exact composition of the military ration then given, but if it were similar to that of the native sailors', the rice in it formed 53 per cent. of the whole.

Van Dieren, discussing Vorderman's difficulty with this episode, puts the same explanation clearly in his own forcible way :

'Is there anything to wonder at? Who ate the biggest ration? Who consumed the least amount of extras? The prisoners every time!'¹

34. *Special Incidence of Beri-beri on Recruits.*—(1) Takaki observed that beri-beri was, of all the soldiery in the Japanese army, especially incident upon *recruits*. He stated—I quote Bentley²—that 'First and second years' recruits were found to be more susceptible to kakké than those in their third year, or older men. This Dr. Takaki considered was due to their wages being smaller and their clothing not so good; and, secondly, to their inexperience of the ways of barrack life, and consequent inability to procure as much food, combined with amusements, with their money, as older hands; in other words, their small wage, their strangeness to town and port life, and a certain amount of ennui, lowered their vitality, and so made them more susceptible to the disease.'

(2) Vorderman makes allusion³ to the special proclivity of this class to the disease among soldiers in the Dutch Native Army—the Pradjoerits, to whom reference was made earlier, (p. 196), were a special instance in point. But in the case of these men—Malays—there was always the sufficient explanation that the rice given them by Government was a different sort from that to which they had previously been accustomed. It was stripped or scoured (stale uncured) instead of unstripped (red or white) *fresh* rice.

(3) In the British Indian Army a similar state of affairs has been noted. In various epidemics among native regiments the disease has attacked recruits in extraordinary disproportion as compared with the 'men.'

For the following details connected with some of these epidemics I am indebted to the principal medical officer, Madras Command, 1903.

During recent years beri-beri has occurred among the native troops stationed at three places only—viz., Vizianagram (successive years, 1896 to 1901), Rangoon (1899, 1900, 1901), and Trincomalee (1901). The troops concerned and the number of cases appear as follows :

¹ 'Kantteekeningen,' p. 28.

² 'Beri-beri—Kakké Gleanings in Japan,' 1893, p. 18.

³ 'Onderzoek,' p. 120.

Station.	Year.	Regiment.	Number of Cases noted.		
			Men.	Recruits.	Total.
<i>Vixianagram</i> : 1 ...	1896	{20th Madras	12	43	55
" 2 ...	1897	Infantry }			
" 3 ...	1898	2nd "	31	42	73
" 4 ...	1899	" "	66	47	113
" 5 ...	1900	(?) "	—	—	11
" 6 ...	1901	(?) "	—	—	15
<i>Rangoon</i> : 7 ...	1899	7th "	—	—	36
" 8 ...	1900	" "	—	—	12
" 9 ...	1901	8th "	—	—	15
<i>Trincomalee</i> : 10 ...	1900	{9th "	—	—	9
" 11 ...	1901	detachment of 138 men }	—	—	36

Unfortunately, the proportion of attacks on men and recruits is not given in the reports on the later epidemics, nor on any occasion the actual strength of recruits and men respectively. But it is sufficiently apparent from the figures as to the earlier outbreaks that recruits did suffer out of all proportion to the men. When it is considered that a regiment consists of eight companies, each of 100 (nominal strength), the total strength being seldom under 600, and that the number of recruits, on the other hand, seldom exceeds more than a few—ten or twenty to each company—it may be seen that the difference in the attack-rate of the disease on men and on recruits was enormous.

For this difference, in some instances, there are noted in the reports of the medical officers dealing with the various epidemics sufficient explanation. But before submitting these, attention should be drawn to the fact that, as mentioned earlier, it was only among Madras regiments that the disease at any time appeared, and to the reason for their special liability.

The daily ration of Indian native troops, substantially the same everywhere and for all arms, is as follows :

Attah, ¹ or rice	2 pounds.
Meat ²	5 ounces.
Dhal ³	2½ "
Ghi ⁴	¾ ounce.
Fresh vegetables	6 ounces.
Various adjuncts ⁵	6½ "

¹ Wheat-flour.

² Mutton or fowl.

³ Pulse, husked and split.

⁴ Native butter.

⁵ Including onions, ½ ounce; tamarinds, 1 ounce; salt, 1 ounce; curry stuff, 9 drachms; pepper, 1 drachm; garlic, ½ drachm.

The native may draw either attah or rice, but by the Madras rice is always preferred. Provisions are at all camps bought locally in the bazaar of the station where the troops are quartered. Hence the rice used is always the same as that consumed, and often grown by, the villagers of the neighbourhood ; and in the case of most of the districts—in the Madras Presidency, at least—this is as much as to say that it is a rice made dry from padi not previously boiled—is, therefore, ‘uncured,’ and more or less old and stale, according to circumstances.

It is in this habit—this addiction to rice—that the Madras troops, the sole sufferers from beri-beri in the Indian army, differ from all the other arms—the Bengal Lancers, Infantry, and Sappers (including the Rajput regiments), the Goorkhas, the Punjab frontier forces (Panjabis, Sikhs, Dogras, Afridis, and Pathans), and the Bombay army of Mahrattas, Jats, and other Sikhs, Rajputs, and Baluchis—for these latter races who do not get beri-beri are almost all eaters of other sorts of grain than rice—wheat, millet, pulses, and corn ; while the few who do eat rice—the Bombay Mohammedans, Deccani Mahrattas—use cured grain.

The quantity of rice allowed daily—2 pounds—is absolutely a very large one. If by any chance the supply were in any way toxic, it seems probable that even the generous allowance of accessories also contained in the diet would not be able to completely nullify its effects. Beri-beri would be likely to appear, therefore, from time to time, among these troops. But the liberal scale of their diet would prevent it from becoming ever as serious a trouble as in the Dutch army or in Malayan prisons.

In regard to the epidemics among the 20th M. I. at Vizianagram in 1896, and 1897, it was noted by Major C. H. Bennett ‘that the disease was most prevalent among recruits,’ and that it appeared among them for the first time only after their arrival at that station.

Surgeon-Lieutenant-Colonel Leapingwell observed ‘that the disease was very prevalent in this district,’ and, further, ‘very few of the cases are married, 43 out of 55 being under three years’ service, most of them being fed in the recruits’ mess.’ The disease is certainly very prevalent in the district, but the 11th M. I. (preceding the 20th at the station) did not suffer in this way to any marked degree. The diet has undergone no change since the regiment was in Secunderabad ; the fact of there being local differences in the condition of the rice supplied at the two stations is, of course, not considered in the statement.

Bateman also remarks—and this is a fact which may be of significance in ultimate beri-beri etiology—that ‘the water-supply this season has been very scanty and bad,’ so that there had been a bad harvest.

In regard to the third epidemic in the 2nd M. I. in 1898 at the same place, the reports reveal some facts of even greater significance : ‘The prevalence of beri-beri among recruits was referred to rice consumed in the recruits’ mess.’ Later it was reported : ‘The issue of a different quality of rice has been followed by a great diminution in the number of cases of beri-beri occurring among the recruits. Since the change from *kutch*a rice to ‘boiled’ rice on September 19, there have been 8 admissions among recruits, as against 17 during the corresponding period of forty days previous to September 19 ; and of the 8 admissions, 5 or 6 contracted the disease previous to the change of diet.’

Referring to the 1898 and 1899 epidemic at Vizianagram, Major Thos. C. Moore wrote : ‘The vast majority of the cases (146 out of 183) occurred between July and October, 1898 and 1899, when the disease assumed epidemic proportions. All the men were meat-eaters to a small extent, as well as rice-eaters. There was no overcrowding, little defect of ventilation. The following improvements have been made in the recruits’ diet—*i.e.*, of the men under three years’ service. “Boiled” has been substituted for *kutch*a rice. *Attah* chupatties in lieu of rice, given twice a week. The ration of mutton has been increased from 6 to 8 ounces daily, except Saturday, when no meat is given, and Mondays, when fowl (4 ounces) is substituted. The *ghee* has lately been increased from $\frac{1}{2}$ to 1 ounce daily. Potatoes, 12 *tolas*—*i.e.*, 4 $\frac{1}{2}$ ounces—have been given twice a week in lieu of other vegetables.’

The proportion of rice in the recruits’ dietary was by these measures considerably lowered, although in all probability it was to the change in the *sort* of rice—from the inferior *kutch*a to the better cured stuff—that the rapid dwindling of the epidemic which followed these measures was mostly due.

Further worth noting, as opposing hypothetical, infective, or miasmatic sources of beri-beri is Colonel Leapingwell’s statement that ‘the recruits occupy separate huts, two men for each hut, distributed through the different companies. They mess in the recruits’ mess separately from the other Sepoys.’

In the next two years very few cases occurred at Vizianagram. Captain R. Bryson, who in 1901 identified the disease as identical

270 THE CAUSE AND PREVENTION OF BERI-BERI

with beri-beri he had seen in China, noted that 11 cases admitted to hospital for 'œdema' were probably beri-beric, and that a similar number had been so described in 1900. Four cases whom he himself treated in 1901, were all fed in the recruits' mess. 'It was suggested by my predecessor that the rice should be obtained from elsewhere, but this was found not practicable.' A 'second' sort of rice was used.

In reference to the epidemic in the detachment at Trincomalee, Lieutenant-Colonel R. A. Quill wrote that 'the barracks occupied by the detachment are ideally constructed and situated buildings. Heretofore they have been invariably occupied by European troops, among whom no case of beri-beri has ever been reported. There is no overcrowding, nor any insalubrity which can be detected.' The diet of the men at that time at Trincomalee was:

Rice	2 pounds.	Turmeric	...	4 drachms.
Dhal	3 ounces.	Vegetable	...	nil.
Ghee	2 "	Meat	...	nil.
Salt	12 drachms.			

For this the following ration was substituted :

Rice	1 pound.	Salt	...	1 ounce.
Attah	1 "	Turmeric	...	$\frac{1}{2}$ "
Dhal	2 ounces.	Vegetable	...	6 ounces.
Ghee	2 "	Meat	...	5 "

No more beri-beri was reported.

At Rangoon, in 1901, the 15 cases which occurred were minutely reported upon by Captain N. P. O'Gorman Lalor, who attributed the origin of the disease to some cause 'endemic in the lines occupied by the 8th Regiment, and which is practically absent from the lines occupied by the 7th M. I. (100 yards only away) and from the surrounding villages.'

It appeared that of the 15 cases 9 were recruits, and 6 only men. As the total strength of all the recruits was but 64, and the men numbered 340, the case-incidence on the two classes was 14.06 per cent. and 1.79 per cent. respectively.

Most of the cases were in Hindus, four companies with a strength of 172 men—nearly all Hindus—giving 11 cases; four other companies—totaling 178 men—who were Mohammedans, only 4 cases. This agrees with many prior observations from the time of Malcolmson downwards, who noted the greater preclivity of Hindus to the disease, and rightly ascribed it to their exclusively vegetable dietary.

Lalor found it significant that the 7th M. I., when occupying in 1899 and 1900 the lines which the 8th M. I. occupied after them in 1901, should also have had beri-beri—36 cases in 1899, 12 cases in 1900 ; but that in 1901, after they had left those lines for others only 100 yards away from the now affected 8th's, they should have been free from it, while their successors there suffered. He postulates some 'place-influence' accordingly. But if there be—and the facts prove there are—certain factors which increase the susceptibility of recruits, and *per contra* reduce the liability of older men to succumb to beri-beri, these conditions alone would afford sufficient explanation of the gradually diminishing incidence of the disease upon an older regiment, and a heavier attack upon new-comers.

The Indian army experience supports, then, the fact elsewhere observed, that beri-beri, when it occurs among soldiers, affects most severely those who are recruits, and that this special liability of the recruit diminishes as his status alters, or as time goes on.

35. *Beri-beri in Siam.*—Dr. J. Campbell Highet, Medical Officer of Health for Bangkok, has favoured me with the following note as to the conditions determining the incidence of the disease there :

'With regard to beri-beri, it is as a rule the poorer classes who suffer, who eat stale rice, not recently milled, and therefore cheaper, and who eat in proportion much more rice, as they can less afford all sorts of adjuncts. *Many of the police recruits suffer from beri-beri within a month or so after joining the force.* They come from up-country, where they have lived in a state of semi-starvation, come down here, get plenty of rice, can afford to buy decent adjuncts, but prefer to spend their odd pence on ice-creams or rice-cakes of all sorts. . . . I have never yet seen a case of beri-beri in the Indian police.'¹

¹ Dr. Highet gives the following number of cases treated in the last three years at Bangkok :

Year.	Cases.	Deaths reported.
1900	1,126	12
1901	1,007	80
1902	2,615	114

He adds : 'In 1890 there was an outbreak in the gaol, which was quickly suppressed. It next broke out during the construction of the Korat railway amongst the contractors' coolies. It was not until 1900 that it commenced to appear again in Bangkok, first in the army, and now it is all over the place and is increasing.'

It seems idle to press the inability of place or infection to explain the peculiar proneness of recruits to beri-beri, for the recorded instances show that an appeal to such possibilities must be futile.

The greater proclivity of recruits, where it is not explained by special differences of rations, may be traced almost always to particular domestic differences affecting their feeding. It is clear that the recruit suffers more than his seniors on an equal ration of the same rice, because :

- (1) He is not used to the *sort* of rice.
- (2) He is unused to so much rice ; the quantity given is in excess of any labourer's or peasant's ration.
- (3) Not being, as a rule, married, he has no dependents with whom his ration must be shared, hence eats it all himself—absolute excess.
- (4) Having to cook for himself, he is less well provided with such adjuncts—fat, oil, condiments, etc.—as domestic cookery supplies—relative excess.
- (5) When supplied with means to buy adjuncts, he wastes them on other things.

The case of the recruit thus falls into line with other noted peculiarities of beri-beri epidemiology—its greater incidence on males than on females, on bachelors rather than the married, on the young and vigorous everywhere rather than the old or feeble. Equally with these, the recruit affords an example of the effect of *relative excess* in the quantity of rice eaten.

36. Beri-beri in the Congo State—an Example of the Effect of Quantity of Rice eaten as Determining the Incidence of the Disease, and of its Reduction as bringing about its Abolition.—The following is translated from the official account given by Bourguignon, Dryepondt, and Firket¹ of the causes of mortality on the Congo during the construction of the railways there :

' During the first years of construction the ration [of the native coolies] was composed of :

Rice	750 grammes.
Mossamedes dried fish	250 „

' These articles were as a rule of satisfactory quality ; the rice was at times broken, but the Medical Department were careful to reject altered and sticky lots, which sometimes were offered. The

¹ ' Congrès National d'Hygiène et de Climatologie Médicale de la Belgique et du Congo, 1897,' 2me partie (Congo), p. 472 (Bruxelles : Hayez, 1898).

chief fault of this ration was its monotony, the coloured labourers finding it very difficult at that time to get fresh provisions by barter in the district where the first works were carried on.

‘ Now, the fearful mortality which prevailed among the coloured men employed on the works in these first years is notorious. Negroes from Barbadoes, Chinese, Guinea-Coast-men fell victims by tens of thousands (*milliers*) to malarial cachexia and beri-beri. In two months of 1892 the death-rate among them rose to 75 per cent. per mille per month, which would have given the unheard-of total of 900 per 1,000 per annum had the conditions persisted. At that juncture the mortality among the coloured was seven or eight times greater than among white men. The latter resisted better because they were better lodged and better fed ; what was wanted, therefore, was to increase the resistance of the blacks. So, to oppose the calamity, drastic alterations were quickly brought about in the conditions of the workmen’s living.

‘ The actual regimen adopted by the railway company for its negro labourers now comprises for the daily ration one fixed component—rice, and four articles varying day by day. Each negro coolie gets daily :

Rice	500 grammes (constant).	
Dried fish	250	”
Salt meat	250	”
Beans	250	”
Delacre biscuits	250	”
					} variable.

‘ These elements are combined so as to have rice, salt meat, and biscuits, or rice, fish, and beans together, etc., the total allowance amounting to 1,000 grammes per head. At certain times 60 to 100 grammes of lard or palm-oil are added, with salt and native pimento (*pili-pili*).

‘ At the same time, the progress of the work, which had at length passed the gorge of Mpozo and the Palaballa ridge, afforded opener ground for camping, and—a matter of importance from the sanitary point of view—the smoother progress gave opportunity to shift camps earlier, so as not to dwell long upon sites where soil became speedily polluted.

‘ The housing of the labourers was improved by getting large waterproof tents, supplied by one of the best makers. Finally, a system of bounties to the men, according to task performed, stimulated their industry, the negroes working harder, and with profit used their extra pay to buy preserved milk, bread, or tinned

European meats. Under the influence of these different conditions, the mortality quickly fell; notwithstanding the dangers inherent in great works of clearing and excavation in the tropics, it came down to about 50 per 1,000. What a contrast with the figures given above,' etc.

These authors deduce from the striking facts which they relate nothing more than a platitude: 'Does not this contrast perfectly show,' they say, 'that the resistance of the negro, like that of the white man, is intimately bound up with different factors of hygienic nature, among which alimentation plays a chief part?' Yet in this bare summary, every line of which breathes the tragedy of ignorant labour sacrificed to more ignorant avarice, so familiar in the tropics, the conclusions of value are plain enough. It has been shown elsewhere that none of those factors of supposed 'hygienic' importance, to the effect of which the causation of nearly all maladies has been at one time or another referred, are of any real influence in determining beri-beri.

Neither heat nor cold, nor soil nor sea; neither exposure to the elements, nor want of air in ill-ventilated dwellings; neither deficiency of food, nor too monotonous a diet, nor 'place,' nor 'contagion,' to every single one of which various influences the disease has actually been ascribed by different observers, is ever responsible for the production of the malady, nor was so in this case.

On the Congo, as elsewhere, when an excess of rice (stale and uncured, 2 pounds of it daily) was eaten, with but $\frac{1}{2}$ pound of salt fish to dilute it, beri-beri 'raged,' as it did in New Caledonia, as it does to-day on Christmas Island, or among occupants of Government gaols and asylums in British and Dutch Malaya. When the quantity of rice eaten was reduced—as it was both absolutely (from 2 pounds to 1 pound daily), and still more relatively, by dilution with 1 pound of other food-stuffs—the malady vanished.

37. Beri-beri confined to Members of one Religion only.—The peculiar limitation (in several recorded epidemics) of beri-beri to natives professing a particular *religion* only, to the exclusion of all others, hitherto unexplained, is similarly accounted for. In instances noted, that of the coolies on the *Ilione*, related by Brémaud (20), and (21) the case of Tamils in the Rangoon hospital, and those quoted from Malcolmsen,¹ it has always been the Mohammedans or Hindus who have been most attacked.

¹ 'A practical essay,' etc.

The former, by their religion, may not, of course, eat pork, the one proteid food also containing much fat which is commonly easily accessible; and the latter may eat neither beef nor pork. They are both naturally, therefore, liable to suffer under circumstances needing considerable dilution of noxious rice with other elements, when pork or beef are the commonest or cheapest, or perhaps the only animal food obtainable. The readier acquisition of the disease by them than by others is thus another example where consumption of rice in quantities relatively larger determines the issue.

38. Other Instances where Predisposing Conditions make Effect of a little Rice relatively Great.—There are other instances in which the amounts of rice taken may remain unchanged, or be absolutely quite small, and normal components of food be taken in proper proportion, and yet the effect be relatively great. The measure of the effect of the poison in such cases is determined rather by other circumstances, various predisposing and favouring conditions, principal among which (after the quantity and proportion in which the poison is absorbed, and the original constitution of the patient), are such agencies as cold, fatigue, and mental depression, prostration through other illnesses, want of fresh air and exercise, and generally all agents which lower vitality.

For the understanding how circumstances such as these may determine or precipitate an attack of beri-beri in individuals whose consumption of the rice producing it may be no more or even less than that of their fellows, alcohol again affords an instructive illustration.

It is a matter of common observation how much more of this poison can be consumed without ill-effect by those who are in active exercise than could be taken by the same persons doing nothing; indeed, by the active generally as a class than by the sedentary; how readily the special effects of alcohol may be produced in the sick, by doses much smaller than would affect them when well; how, above all, not in the chronically alcoholic only, but in those who may be for the time only under the influence of alcohol, a simple accident or surgical injury is prone to set up delirium tremens.

The principal illustrations of this increased relative effect of ordinary quantities of rice are to be seen in what may be called 'surgical' beri-beri, and the beri-beri of the lying-in.

39. Surgical Beri-beri.—It is common enough in the hospitals of the East for patients admitted for quite other complaints to

acquire, sometimes even to die, of beri-beri in the wards. This seems to have been noticed to occur oftener—possibly because of the contrast, possibly because, being actually less ill, they eat more than other patients—in patients with quite trivial ailments; and—again, possibly because the usually less severe condition of such patients renders the beri-beri more obvious, and their appetites are also better—in surgical rather than medical cases. Some of these, doubtless, are merely 'larval' cases—patients in whom the disease was on the point of breaking out, and in whom it must have declared itself, wherever they happened to be, shortly. Yet others, it is painful to think, must owe their attack at times directly to the food supplied them in the hospital itself. For it must happen that hospitals, equally with other institutions and the public, sometimes receive and distribute the same rice with its invisible poison which operates to produce beri-beri elsewhere.

But more of these cases represent, without doubt, a conjunction of lowered resistance with the application of an amount of toxic influence such as, though ordinarily innocuous, is, under the circumstances, effective enough to determine beri-beri. Yet others, it may be conceived, are, like chronic alcoholic subjects, beri-beric to a degree which remains tolerable, and unsymptomized under normal conditions, yet in a state of what may be termed toxic strain, the breaking-point of which is reached upon injury, the stress of which takes the form whether of nerve-shock or the action of toxins absorbed from wounds. Jefferson¹ has given a graphic account of the sudden supervention of this dreaded malady in fatal form after quite trivial operations, as exemplified by cases under his care in the Philippines.

Reaucar² draws attention to the liability of robust and apparently healthy persons to succumb unexpectedly to beri-beri after quite insignificant operations. 'We have witnessed,' he says, 'many instances of the kind. There dwells yet in our memory the case of a young Cambodian who presented himself on the morning round to get a tooth extracted which had pained him during the night. It was a first molar, with a small carious spot. This young man was robust, and his general state of health excellent. So we proceeded to extract the tooth, which came out very easily. But our Cambodian was seized the same evening with symptoms of acute beri-beri, and died of it that very night.'

¹ *British Medical Journal*, 1898, vol. i., p. 1257.

² 'Le Béri-béri à Poulo-Condore,' p. 50.

Reaucar relates other such instances, and dwells particularly on the disproportionate incidence of the disease on patients having only slight wounds, as ulcers of the foot—a common ailment. When such ulcers remained long intractable to treatment, he was led to see in their condition a sign—the only one discoverable—of the presence in ‘larval’ form of the feared disorder: ‘Alas! when we perceived such an ulcer, “immovable in space and time,” we knew what fate to expect for the patient. He was classed in the list of beri-berics to be.’

The writer recalls—and every Eastern practitioner must do the same—many an operation in which the fair prospects of the patient ended in such unforeseen catastrophe. And although now that in the writer’s hospitals no rice except cured rice is given to patients, such results do not occur, he yet views with apprehension the event in any case of operation upon a patient who previous to it (before admission to the wards) had been long upon the beri-beri-producing dietary.

In the Medical Report of the Straits Settlements for 1904 mention is made of the fact that five patients (among 3,161) admitted for other complaints acquired beri-beri in the *General Hospital*, Singapore, and no less than 60 (among 5,411 patients admitted for other diseases) in the Tan Tock Seng, or *Pauper Hospital*. In 1891 10 per cent. of the patients were so attacked, and one-third of them died.

40. **Puerperal Beri-beri.**—Much more serious is the frequency and gravity with which beri-beri attacks the lying-in. In the Philippines especially this phase of its epidemics has been a veritable scourge. It is also frequent among Eurasians and Babas, as well as native Portuguese in British Malaya. Among the latter classes it is the invariable custom to feed their women during the puerperium on little else than messes and gruels made from rice; and cases which the writer has seen have been fed entirely on such diet for weeks after the birth. Is it surprising that, at a time when resistance is reduced so largely, the deprivation of proper nutrient food and relative great increase in the amount of rice consumed should produce beri-beri?

It is a confirmation of the explanation of the mode of production of these cases which has been given, that they immediately yield to the simple remedy of substituting for the uncured rice, which is invariably being eaten, some other sort, or, preferably, a wholly different food.

Takaki ascribed to similar conditions the severe incidence of

beri-beri on women after childbirth in Japan. I quote from Bentley¹ the following :

'When asked how he accounted for the fact of the disease attacking women after childbirth, and seldom, if ever, at other times, Dr. Takaki stated that in Japan it is the custom for women to forego all nitrogenous food, such as meat, fish, eels, etc., almost from the time of conception to recovery after delivery. This he considers so reduces or alters the blood's character as to bring about changes which induce beri-beri, and in substantiation of his opinion he stated that in his own practice, where from the first he feeds his patients well with barley, meat, etc., in addition to rice, he has not for years seen a case occur after parturition in his extensive practice.'

* * * * *

The literature of beri-beri abounds with many and striking instances similar to these cited, but enough have been given to prove the point—the variation of beri-beri with quantity of rice consumed.

Experience shows, what was *a priori* to be expected, that no *absolute* scale of toxic dose or quantity of rice can be fixed. The first and chief determining factor is the degree or intensity of the poison in the sample eaten, and this at present is indeterminable, except through its effects. But this much of sound inference the facts seem to afford : namely, that so long as the daily amount of rice taken does not exceed 14 ounces absolutely, or form more than 33 per cent. of the whole diet, no beri-beri will appear in the healthy ; although in the case of the sick it may be supposed smaller doses might set up the disease.

Looking to the conclusions, which the whole body of facts affords, it must be admitted that the evidence, brought together from every quarter, including some phenomena long and otherwise inexplicable in the epidemiology of beri-beri, shows that it is to excess of rice consumption, whether absolute or relative, that the malady is due.

* * * * *

The third among the deductions from the rice theory must thus be regarded as proved : *that the incidence of beri-beri is directly proportionate to the quantity, whether absolute or relative, of rice eaten.*

¹ 'Beri-beri—Kakké Gleanings in Japan,' p. i.

SECTION IV (*continued*)

BERI-BERI PERSISTS WHILE USE OF SAME RICE ON WHICH IT WAS ACQUIRED IS CONTINUED; CEASES WHEN THIS IS DISCONTINUED

IV

SUMMARY ; LIGHT THROWN ON OBSCURE FEATURES OF EPIDEMIOLOGY BY PROOF THAT BERI-BERI IS A GRAIN-INTOXICATION

Fourth Deduction.—Last among the deductions from the rice theory, which could be derived from no other premises, was the following :

That the extent of epidemics—the number of persons attacked in a given community, when all are not affected simultaneously—should persistingly increase so long as the same rice should continue to be used ; and that the course of the disease in those affected should not tend, or tend little, to recovery, so long as they should continue to use the same rice as when they acquired beri-beri ; and, conversely, that it should so tend, if not invariably, at least more often than not, to recovery, when the old rice should be discontinued ; or, briefly, *as dependent merely upon the sort of rice eaten, beri-beri will persist as long as the same rice is used, and disappear when this is discontinued.*

The evidence upon which the demonstration of this truth depends is abundant, but much of it has always been, through a very obvious fallacy, presented under quite another aspect ; and, as a matter of fact, changes in the condition of beri-berics and in epidemics of beri-beri, which have been really due to alteration in food, have been almost invariably ascribed to change of *place*.

It is easy to understand how those who have not understood, or have discredited the influence of the rice factor in beri-beri have failed also to appreciate, or to note, the actual facts in

regard to change of their diet by patients who at the same time have also changed their *place*; how, with attention narrowed to the perception only of the expected place-factor, they should have ascribed to it alone results properly attributable to the less apparent one of food. But, obviously, in considering the effect upon an epidemic or upon cases of movement from one to another locality, more than the mere fact of transfer has to be taken into account. With the change of place other changes occur, and among these a nearly constant one is alteration in the source of food-supply. In the case of beri-beri no general change in style or kind of diet even is necessary to bring about important differences in the results. Alteration in the source, the sort, or the parcel merely of the market-supply of the same sample of rice eaten, may make all the difference; and who shall say, in any given instance of change of locality, to which may perhaps have been attributed the cure of a patient, or the stamping out of an epidemic, but in the narration of which the part played by food has not been considered, that there was no such accompanying alteration in this factor also?

In point of fact, when dealing with persons whose diet is, in any event, rice of some kind, it is impossible to assert in any instance that there was no alteration in the rice at a given time, since the poisonous principle in it is not yet recognised, so that a statement in regard to any epidemic that fluctuations in its incidence occurred—whether accompanied by change of place or not—*without* any alteration in the rice eaten are of little value.

But in most cases, in which improvement is ascribed to change of locality, definite alterations in the food-supply are to be observed if they are looked for. This is the writer's own experience, and it applies equally to published records.

Dutch East Indian Fleet Experiences.—Thus, Van Leent,¹ recounting his own experiences in the Dutch East Indian Fleet from 1870-1878, says: 'During the first three years the vessels were stationed off Muntok in Banka, off the south-west coast of Borneo, and on this station beri-beri among the native crews, and even the Europeans, was constant; but it was observed that whenever the same vessels went to Palembang the disease disappeared.

'Those,' says Van Leent, 'who favoured the endemic origin of beri-beri always relied upon the fact that beri-beri vanished as by magic whenever a ship on which the disease prevailed

¹ *Gen. Tijdschr. v. Ned.-Ind.*, xx., 5 (1880), p. 297.

SECTION IV (*continued*)

BERI-BERI PERSISTS WHILE USE OF SAME RICE ON WHICH IT WAS ACQUIRED IS CONTINUED; CEASES WHEN THIS IS DISCONTINUED

IV

SUMMARY ; LIGHT THROWN ON OBSCURE FEATURES OF EPIDEMIOLOGY BY PROOF THAT BERI-BERI IS A GRAIN-INTOXICATION

Fourth Deduction.—Last among the deductions from the rice theory, which could be derived from no other premises, was the following :

That the extent of epidemics—the number of persons attacked in a given community, when all are not affected simultaneously—should persistingly increase so long as the same rice should continue to be used ; and that the course of the disease in those affected should not tend, or tend little, to recovery, so long as they should continue to use the same rice as when they acquired beri-beri ; and, conversely, that it should so tend, if not invariably, at least more often than not, to recovery, when the old rice should be discontinued ; or, briefly, *as dependent merely upon the sort of rice eaten, beri-beri will persist as long as the same rice is used, and disappear when this is discontinued.*

The evidence upon which the demonstration of this truth depends is abundant, but much of it has always been, through a very obvious fallacy, presented under quite another aspect ; and, as a matter of fact, changes in the condition of beri-berics and in epidemics of beri-beri, which have been really due to alteration in food, have been almost invariably ascribed to change of place.

It is easy to understand how those who have not understood, or have discredited the influence of the rice factor in beri-beri have failed also to appreciate, or to note, the actual facts in

Klosser's intent in this relation was to show the dependence of the disease on salt fish, since it so happened that it was only when this commodity was being eaten, or much eaten, that beri-beri occurred. Van Dieren cites the case as illustrating the influence of bad rice, upon which, as elsewhere, it did undoubtedly depend: the rice in use, when the disease broke out, being both of an uncured sort, and unusually stale, and also eaten, as compared with other times, relatively in great excess.

The purpose which the incident serves in this section is, however, to illustrate the point—the fallacious point—which such a history gives to the conception of the influence of locality. The ship while engaged in one locality, before 1885, had no beri-beri. During that year, cruising elsewhere, the disease appeared—to be reduced by appropriate feeding. But *after having returned to Onrust*—a new locality—it reappeared. In this case no such fallacy as the dependence of the disease on place, or upon a mysterious *noxa* implanted in the ship itself, was postulated by the observers. But how often has the contrary been the case!

How easily the truth may remain concealed, even when diligently sought, the following instance will serve to show:

An Epidemy in Jelebu.—Sixty sinkhehs were imported at the New Year in 1900 to work at a mine in Jelebu. Within six months twenty-six of them had beri-beri—some died. Consulted by the manager, a European, anxious to do everything possible to stay the plague, I visited the place. As is usual, nothing was apparent which could afford explanation of the epidemy. The disease affected, indeed, only the sinkhehs, who occupied a separate building by themselves; but this was contiguous to sheds in every respect similar, the occupants of which (laukhehs) were not attacked, although between them and the sinkhehs there was frequent and close intercourse. The sinkhehs' food was provided for them by the employer, through a local contractor, and in quality and quantity appeared to be good. The laukhehs supplied themselves. At that time I had not gained that conviction of the importance of the difference between the laukheh and the sinkheh in regard to feeding which has since become impressed upon me, but I believed that the essential element in beri-beri-causation lay in uncured rice, and supposed that some factor of acquired tolerance must be, in this and other such cases, the cause of the exemption of the laukhehs, whose rice was of the same sort as that eaten by the sinkhehs. I accordingly advised the manager to pro-

vide a new diet for the latter, the rice entering into which should be, if possible, of the cured kind, or at any rate, some sample, which its consumption without harm by others should have proved to be innocuous, and that the nitrogenous elements of the diet should be increased. At the same time the benefits owned to have ensued upon removal of the sick to fresh localities, in many instances, were also recounted, and the recommendation made that entirely new quarters should be provided for the sinkhehs on a new site.

The latter part of this advice only was followed. A new shed was built, not a hundred yards from the old one, which was burnt down, and the sinkhehs moved into it. The suggested alterations as to diet were not carried out, as the manager informed me he had found them impracticable.

Nevertheless, immediately the new building had been occupied the sickly began to improve, and not a single fresh case of beri-beri occurred there.

The epidemic was brought to an end by nothing else, apparently, than mere change of habitation. Topically, even the site of the abode as well as the scene of the labours of these coolies, remained the same, as did also all the other accidents of their environment.

In spite of the difficulty which militates against such an explanation of this and all similar instances, namely, of understanding how an infective agent, capable of continuously operating upon all the inmates of one building, and known, through other evidence, to be of a transportable nature, should have failed to become in some part transferred to their new abode by a mob of coolies, among whom its effects were yet active and recent, and to whose persons and belongings no sort of disinfection was applied—it seemed impossible to find for the problem of beri-beri-causation in this Jelevu instance any other solution than that of ‘place-infection’—a miasm—confined to the actual building in which the cases occurred.

I must confess that this particular case long formed an insuperable obstacle in my own mind to that complete demonstration of the causation of beri-beri by rice to the truth of which so much other evidence seemed to point.

It was only when, a year afterwards, I was discussing the question with the same manager, and mentioned the stumbling-block which the case of his coolies offered to the rice theory, that the real explanation of the event appeared. He said that it

was true that he had found it impossible to arrange to give the sinkhehs the different diet which had been advised, but, as they were near the end of their term of engagement as sinkhehs, he decided to cancel the remainder of their contract, giving them their freedom and the wages of free coolies. They then were no longer fed by the contractor, but bought their own provisions, like the other laukhehs, in the market.

The most striking piece of evidence which I had met with for 'place-infection' became thus one of the common examples of dependence of beri-beri upon food. Not only did the freed sinkhehs become enabled, as laukhehs, to earn more wages and provide for themselves a more varied diet, thus reducing, at any rate, the relative quantity of rice eaten, but it is certain that the village supplies, guarded by competition, were of better rice than would be found for them by a contractor for rations at a fixed price per head.

This phenomenon, the effect of change from relatively much to less rice in the dietary, without any change of place, in reducing or eradicating beri-beri, is evidenced by thousands of instances every year in British Malaya, *when sinkhehs become laukhehs*.

Recoveries following Change of Place.—It is unnecessary here to multiply instances to show how rapid and striking usually is the improvement which follows the removal of the patient from the place (involving, it is to be postulated, always a different diet) in which the disease was acquired. This feature of beri-beri is accepted and commented upon by most observers. It happens often, strangely enough, even when the district to which the sick may be removed is itself one in which the disorder is 'endemic.' On the other hand, the expected benefit does not always occur, even when the new locality is free of all taint of endemicity. Such inconsistencies, inexplicable so long as the cause is conceived to be a strict place-emanation or infection, are readily to be understood when referred to their true origin in food.

The influence which change of 'environment' exerts, being essentially due to change of diet, it is through alterations in the latter factor (whether visible or not) that the amelioration of cases is brought about.

It is to be remembered that it flows from—is even a necessary result of—the rice theory, that not all rice—not all parcels even of the same market-sample of rice—shall be equally poisonous. It is also probable that the sort of rice which usually contains

the poison is *oftener free from it* (i.e., in toxic proportion) *than not*, otherwise beri-beri would be almost constant and universal. It results, therefore (on the mere theory of probabilities), that the sufferers from a bad sample, changing their locality and food-supply, are most likely to find their next rice innocuous. They may find this so in a district other occupants of which are consuming perhaps a noxious stock, and consequently getting beri-beri. They may be unfortunate enough against the chances to be supplied with bad rice where the portion allotted to others is good. These are the natural results flowing from a poisoning, the agent of which is unrecognisable and irregularly distributed. They are features shared by all epidemic poisonings through food.

How little the factor of change of place (and food) is to be *depended* upon in securing benefit to patients the history of the beri-beri hospital at Bagan Pinang, in Negri Semblian, shows. Built by the Government in 1896, on the recommendation of the writer—before he had attained his present convictions—to secure for all the beri-beric patients of the State who could be removed there the advantages which any change of locality, but especially removal to the seaside, was believed to bring: this hospital, when first occupied, seemed to justify expectations. In 1896, 1897, and 1898, the death-rate was lower, and the rate of recovery more rapid than in other hospitals in preceding years. In 1899 and 1900 these conditions were reversed. In 1901 and 1902 the former conditions were repeated.

These variations in mortality, extensive examination of statistics has shown, were not peculiar to Bagan Pinang. Similar fluctuations took place at the same periods throughout the States.

Of the benefit which attends removal of *patients* from their homes to hospitals generally—the extent or reality of which is problematical—apart from nursing, much, if not most, therefore, must depend on the chance of their getting in their new quarters rice (or other food) less noxious than they had been consuming. It is true that in acute stages of the malady seldom much nourishment of any kind can be taken, and what is given is something more nutritious and more easily assimilable than rice. But no sooner does recovery begin than the patient clamours for rice, and in some form or another usually gets it, with the not infrequent result of a 'relapse.'

Since the rice used in these institutions¹ is also of an uncured

¹ I regret to say still is (in January, 1906) in all local hospitals, save those under the writer's charge.

sort—Siam or (and generally) Rangoon, the cheapest—it is manifest that it is quite a chance whether the sample in use at any time may not be quite as poisonous as any which may have been in consumption outside. The case of such patients will, then, not be improved, but aggravated, their last state become worse than their first, by so much as it would be better for them to be starved of all rice without, than carefully treated with a poisonous sort within, the hospital.

That these conditions are at times actually realized is more than probable. The appalling case-mortality which in some years or seasons happens at hospitals at which at other periods the rates are even very low, supports this conclusion; while the occurrence of primary attacks in patients admitted to the wards for quite other complaints has only too often had to be deplored.

But, if the benefits attending change of place in all cases predicate and are explained by simultaneous change of food-supply, no less is it to be assumed that in places where beri-beri persists—unless, the point having been expressly investigated, the contrary can be asserted—the kind of rice eaten remains the same.

The tendency of patients, left in the environment in which they acquired the disease, to get progressively worse—a feature not characterizing any disorder known to be dependent on infection—is a matter within the experience of all who are well acquainted with its epidemiology.

It might seem, upon this showing, that all cases whose circumstances so remain unchanged should die; and where there really does occur no intermission in the application of the poison it is likely that most do so. That this does not often happen is due, no doubt, partly to the fact that the supply and degree of intensity of the toxic principle in the rice are liable to variation, partly to the automatic operation of the natural factor, that the very prostration induced by the disease prevents them from taking more of the food which caused it; in greater part still to the acquisition of some degree of toleration, as in the case of so many other poisons.

But if the reputed influence of place over beri-beri is, in most cases, a fallacy which has resulted from the assumption that there has been in them no alteration in the food-supply, there are accounts of two epidemics at least in which it has been claimed that among persons purposely made to share exactly

the same diet the incidence of the disease has been determined merely by difference in location.

These are the experimental observations made by Travers and H. Wright respectively in the Selangor Gaols.

Travers' Experiment at Pudo, 1895.—Travers,¹ who experimented in 1895 (but whose results were only published in 1902), sought to determine the influence of the food factor by feeding two bodies of prisoners for some ten months upon identical diet, but at two different places—one of them an old prison, reputed to be, and to have always been, incapable of originating beri-beri; the other a new gaol, at which beri-beri was then, and has since continuously been, severely prevalent.

There must be recalled here the conclusion, submitted earlier in the work, that beri-beri is, at all events in most cases, a disease which takes, not days, but months to produce. Numerous instances which were cited went to show that the ordinary period of 'incubation' is about seven months.

In an experiment such as Travers made, therefore, it was essential that all the individuals taken for comparison should be kept under observation for at least this period; and elementary, that the terms served by members of each of his two parties should be equal.

But no steps were taken to secure either of these conditions.

Instead, it appeared that all those most likely to get the disease—namely, the long-sentenced prisoners—were kept at the one prison, where most of them acquired the malady. The result being that which might have been expected—namely, that no cases of beri-beri occurred at the other prison—Travers, whose notice this source of fallacy entirely escaped, concluded that rice or food played no part in the production of the disease.²

H. Wright's Experiment at Pudo involved in same Fallacy.—H. Wright,³ working at the same prison later (1901-1902), who might have profited by the experience of Travers, unfortunately repeated his mistakes.

The following is an account of his observations:

The material for experiment was the whole body of prisoners incarcerated in Pudo Gaol.

¹ *Journal of Tropical Medicine*, August 1, 1902.

² Cf. *Travers' experiment*, Appendix. A detailed examination is made in the Appendix proving the points here stated.

³ 'Studies from the Institute of Medical Research,' vol. ii., No. 1. 'An Enquiry,' etc.

The number of inmates when the experiment began was 385.

The daily average was between 285 and 530, the mean being 458.

Each prisoner occupied a separate cell, 'satisfactory as regards size.'

The observation extended over eleven months, from May 3, 1901, to April 1, 1902. Throughout this period a similar special 'physiologically correct' diet, containing an unvarying quantity of 21 ounces of rice,¹ was supplied daily to all.

'The rice used was a good quality of Siam or (*sic*) Rangoon.'

The prisoners were divided into four groups or parties. In the first and fourth were placed all known or doubtful beriberics. In the second and third only those 'who did not then have, and never had had, beriberi.'

These parties were kept entirely distinct from each other, occupying different blocks of cells at night, and working in different sheds by day. Party 3, however, did not work in the sheds. Their work was 'wholly extra-mural' (para. 277, p. 42). Their only communication with each other or the outside was through the intermediation of the beriberi-free Party 3, who were used as carriers. All subsequent admissions were assigned, if already beriberic, or with a doubtful history, to Party No. 1; if entirely free from it, to No. 2.

Under these circumstances it happened that beriberi became manifested in one form or another in three of the four parties, namely, the first, second, and fourth, but not in the third.

Having, as he supposed, made not only the food-supply, but all the conditions (as to clothing, sleep, water-supply, personal hygiene, etc.) as uniform as possible to all parties, Wright concluded that food could have had no share in the causation of the disease, and that it must have been due to local infection. 'Any assumption,' he states, in summing up his results, 'that the diet of the prisoners, looked at qualitatively or quantitatively, was a factor in the production of beriberi in the gaol, is contradicted by the entire escape of members of Party No. 3, who were on the same diet as Parties Nos. 1 and 2 [and also No. 4].' ('Study,' par. 357, p. 57).

Now, had the circumstances of all the parties been, as Wright considers they were, precisely equal, 'the entire escape' of one of them from a disease which attacked the rest would undoubtedly have afforded a strong presumption that the cause of it could not lie in a factor—food—which they had in common; but not

¹ Together with 6 ounces of meat, 9 ounces of vegetables, 1 ounce of fat (cocoanut-oil, 1½ ounces), curry-stuff, and salt.

even this would be the complete proof which Wright claims, since there is such a thing as difference in personal susceptibility—not all become intoxicated by a dose of alcohol which sends, perhaps, some under the table; nor do all those get enteric who eat civic oysters at Colchester.

It is fortunate that in Wright's instance we have more than a mere general statement of the results, partial presentation of which renders fallacies escaping the original observer so difficult to expose.

But his account of his observations is far from complete. Facts vitally important to comprehension of the real relations of beri-beri to his prisoners are omitted.

For instance, there are given analyses of the cases of beri-beri occurring in the prison according to age (Table 27); general health (Table 28); relation to time of the year and to meteorological conditions, to term of incarceration, and to occupation before admission (Tables 29, 32, and 34); but no indication is given of the relation to these factors of *other prisoners* in the gaol at the same time who had not got beri-beri, comparison with whom (by means of similar analyses) alone could have given the tables any value. The personal record of each case—at least, so far as length of sentence and time served before, or without getting beri-beri—could easily have been supplied in regard to every admission to the prison during the eleven months, and would have been worth the whole pamphlet, but nothing of the kind is furnished. It is not even possible to gather *how many individuals* absolutely entered into the composition of the parties, save one, the incidence of beri-beri upon which was to serve for a conclusion about its origin so important as that which was announced.

How little that conclusion is justified, even by his own facts, Wright's own statements will now be adduced to show.

Authorities are agreed that the incubation period of beri-beri is indefinite. It has been shown earlier that the period is variable, but generally very long, and how cases in which it appears short may be explained as those of which observation has covered only the later stages. The process is thus to be conceived as one of slow poisoning, the effect of which is accumulative and unmarked by any signs, until it reaches the point of intensity (necessarily varying in different individuals) at which the resistance of the organism breaks down.

So long as a theory has validity—and this must be granted to the present one—which no facts oppose, and for which several recognised chains of causation supply complete analogy—namely, the paralyses resulting from lead, from arsenic, and if not the peripheral, at least the central results of alcohol—it has to be reckoned with, and little value can attach to researches in which it is ignored.

Wright, whose peculiar reasoning appears not to have permitted him to conceive any other manner of causation of the disease save than by 'infection,' arbitrarily assumed that there was a period of incubation of all cases of beri-beri, and that this was 'between ten and fifteen days.'¹

In this belief he disregarded entirely the possible influence of *time* in determining the incidence of the disease upon his prisoners. In the formation of his parties for observation, their members, whether long sentences or short sentences, whether they were to be exposed to the influences causing beri-beri in the gaol for a day only or for a year, were treated as units of equal value.

Yet this, of course, they were not. Even were beri-beri produced by infection (as Wright thinks it is), it should have been obvious that the longer a prisoner should remain in gaol, the more chance he would have of becoming infected.

One of the few incontestable facts which these gaol epidemics have demonstrated is that the liability to get the disease increases with the length of exposure to the cause—*i.e.*, in gaol, the length of time served. It will be recollected that it was shown² that the incidence upon long-sentenced prisoners at Pudoh was in 1896 and 1897 *more than total*—many cases getting relapses—and in 1902 not less than 83 *per cent. for primary attacks*, as opposed to an incidence upon short-sentenced prisoners in the same years, never exceeding 3 per cent. Also that long-sentenced prisoners formed altogether only 5 per cent. of the total committals at Pudoh.

Such enormous sources of disparity between the two classes of prisoners make it a fallacy to regard them as equal. It is manifest that the inclusion of more or fewer long-sentenced men in one party would make all the difference in the liability of that party to beri-beri. And since, in Wright's experiments, this factor was ignored, his conclusions become on that account alone unreliable.

But this, though sufficient, is not the sole reason for rejecting Wright's results. The conclusion that beri-beri could not be due to food was based upon the exemption from it merely of one party. References to his pages will show that on the ground of numerical probability alone, *it was to be expected that that party should so escape.*

¹ *Loc. cit.*, p. 10, par. 84: 'I have no hesitation in saying that the incubation period of beri-beri is between ten and fifteen days.'

² P. 77.

VARIES WITH VARIANCE IN RICE-SUPPLY 291

At the beginning of the experiment there were 285 prisoners in the gaol. These were divided as follows :

Party No. 1.	Party No. 2.	Party No. 3.	Party No. 4.	Total.
101	113	33	38	285

All subsequent admissions were placed in either Party 1 or 2.

' Those with signs or history of having had the disease were allotted to Party No. 1, those free from beri-beri to Parties 2 or 3 ' (par. 279) ; but—

' Party No. 3 was composed of thirty-three prisoners who at the time did not have, and never had had, beri-beri. It was placed in the cells of the administration block to sleep. It should be stated here that no case of beri-beri is known to have originated in this block. The work of Party No. 3 was wholly extra-mural. . . . This party averaged about thirty-six during the eleven months' observation. *Its strength was maintained by allotting to it prisoners from Party No. 2, who, on incarceration, were found to be free of signs and history of having had beri-beri* ' (par. 277). The italics are the present writer's.

Par. 278 : ' Party No. 4 was composed of about thirty-eight cooks, dhobies, and tailors, who had had more or less severe beri-beri in the gaol, but who had almost wholly recovered. I was, in view of their having had beri-beri, warranted in regarding them to be to a large extent immune.¹ This party scarcely changed at all during the eleven months' observation, all of its members being long-sentenced prisoners.'

Wright's table (20) shows that the average daily strengths of Parties 3 and 4 were maintained at thirty-four and forty-one persons exactly during eleven months of observation. The strength of the others varied slightly. They appear as under :

Actual Daily Average Strength of Parties.

No. 1.	No. 2.	No. 3.	No. 4.	Hospital.	Total.
129	222	34	41	32	428

¹ A view which nothing justified, and which the fact (admitted in par. 332) that men in this very party also showed signs of relapse proves was unsound.

292 THE CAUSE AND PREVENTION OF BERI-BERI

The proportional strength of the parties per cent. of the whole was :

No. 1.	No. 2.	No. 3.	No. 4.	Hospital.	Total.
28	48	8	9	7	100

The strength of Party No. 2 was, therefore, six times as great as that of Party No. 3, and the odds against any prisoner who was about to get beri-beri being admitted into the latter were at least in that proportion; and when to this consideration is added the fact, that of all the healthy prisoners who came in during the period only 6·4 per cent. got the disease altogether, it is seen that the chance against any member of Party 3 being one of those cases was 90 to 1.

This chance was further diminished by the fact that the additions made to Party No. 3 to keep up its strength were not unselected fresh admissions to the prison.

‘ Its strength was maintained by allotting to it prisoners from Party No. 2.’

That no prisoners were passed directly on admission to Party No. 3 is made quite clear by par. 338, which states that *all prisoners admitted to gaol free of beri-beri* during the period of observation were ‘ immediately allotted to Party No. 2.’

Yet again, the chance of beri-beri occurring in Party 3 was further reduced by a special selection of the members assigned to it, resulting from the fact that ‘ Party No. 2 was further protected by the immediate transference to Party No. 1 of any member of it who developed acute beri-beri ’ (par. 279).

Last, the consideration already alluded to, that it is chiefly upon long-sentenced prisoners that the incidence of the disease falls, renders the probability that any prisoners admitted to this party—No. 3—should have acquired beri-beri, so much the more exiguous, that it might have been expected to be—what the result showed it was—*nil*. For analysis of the prison records shows that less than 5 per cent. of all prisoners committed in any year and less than half the daily strength are long sentences.¹

¹ It is interesting to note that among 1,406 prisoners who were passed into Party No. 2 as free of beri-beri, 90 subsequently developed the disease, or 64 per 1,000, among whom were three deaths, or 2 per 1,000 of the population, rates which correspond fairly with those adduced earlier in this work as prevalent among the population generally.

Wright relates, in further disproof of the view that beri-beri is attributable to rice, that 'the rice supplied to prisoners was absolutely sterile.' He lays stress on the fact that '*the rice before being delivered to the prisoners for their midday and evening meals was steamed for two and a half hours, under two atmospheres of pressure ; while that for the morning meal was boiled from 8 p.m. to 5 a.m. to form congee, or porridge—a treatment sufficient to exterminate all known organisms and destroy all toxins,*' and, it may be added, to bring into solution or emulsion any poison little soluble which might be present.

I do not know Wright's authority for stating that *all* toxins would be destroyed by this treatment. But it is not necessary to conceive of the beri-beri-producing principle in rice as being a *toxin*. It may be—in greater likelihood is—an alkaloid, or a compound like ergot, which is sufficiently poisonous, and is not destroyed by boiling, and of which such treatment might even favour the extraction.

So far, then, the evidence which shall connect beri-beri strictly with *place*, the food remaining unchanged, is yet to seek ; while the presumption remains justified that in all the instances in which influence upon the disease has been ascribed to change of locality, the attribution has been a fallacy, the real factor being always food.

The inference thus made would be strengthened into a certainty could any series of cases be brought forward in which the spread of beri-beri in communities, or its course in individuals, should be shown to have become arrested, when the food in use only was changed, whether in kind or as regards quantity of rice eaten, the place and other circumstances remaining the same.

The accounts of occasions upon which such change of food alone has determined the disappearance of the disease are numerous and unequivocal.

1. In Individuals, according to the Writer's Observations, this always happens.—In *individuals it is the writer's constant experience that the course of the disease is checked at once, in mild and subacute cases, merely upon changing the sort of rice eaten, without the application of any kind of 'remedy,' and without alteration in any other circumstances.*

The same result ensues, necessarily more slowly, but with equal certainty, in even grave cases, provided the lesions induced by the malady are not already irreparable.

In all cases in which it has been possible to gain a clear account

of the progress of their condition from the patients themselves, as in the case of Europeans, Eurasians, and Malays,¹ it was *uniformly noted that the relief of symptoms became noticeable within the first day of abandonment of the injurious food*; in especial, the vague sense of discomfort or uneasiness, of oppression of breathing, or depression of spirits, of weariness, and of fatigue or even breathlessness after slight effort, which mark the onset of the disease, disappeared rapidly. Such patients professed themselves as *feeling* quite well, even before such signs as shin-œdema, and anæsthesia had vanished.

It is unnecessary to cumber this work with details of such cases, notes of many of which could be given. It will be sufficient for the writer to urge the result of his own experience—that change of the rice eaten is the first and only remedy necessary to be prescribed for beri-beri patients in practice. It is the one and only remedy upon the curative effect of which an absolute reliance may be placed.²

2. **On the Ship 'Ancona,'** it will be remembered, of four persons attacked in one mess the two who changed their cabins, but continued to eat the same food, died; the two who kept to their cabins, but entirely altered their diet—excluding, among other things, rice—recovered.

Of instances in which the arrest of *epidemics* has followed change in the sort of rice eaten there is no lack. Some of them have already been mentioned in this work. The subject is one of such importance, however, as to justify their recapitulation.

3. **The Indian Marine Survey Cases—Anderson.**—In the epidemic described by Anderson in the ships of the Indian Marine Survey we have it on his authority that '*all other measures failed*' to check the disease, *until all the old stock of food was 'hove overboard; a fresh supply was obtained from Karachi, and the disease at once ceased.'* While this happened on the *Investigator* the disease was continuing among the members of a detached boat-party, and in her consort the *Nancowry*. But when these crews were put on the fresh provisions the disease ceased in them also.

4. **The Pearling Fleet—Haynes.**—The experience of Haynes, derived from seven voyages on pearling schooners, in all of which beri-beri severely attacked the crews, was to the same effect.

¹ The fog of language and the extraordinary pathological conceptions of Chinese and Tamils make the interpretation of their meaning very difficult.

² *I.e.*, in any but pernicious or already moribund cases, and provided that the new rice allowed is 'cured' or 'fresh.'

The substitution of other articles of food or of fresh rice for that upon which the men had been fed at once arrested the disease.

5. **The Malacca Training College.**—At the Malacca Training College resort to 'cured' instead of uncured rice brought about cessation of the epidemic.

6. **Fresh and Stale Rice in Philippine Prisons.**—An important instance is narrated by Littlefield.¹ He states that at Lingayen, Pangasinan, beri-beri was prevalent in the prisons and hospitals, the rice used being Chinese white rice supplied by the Commissariat. This was exchanged for the sort grown and consumed by the natives locally, and thereupon beri-beri disappeared. In the civil prison, he says, although the sanitary conditions were worse than in the other, the *local* rice had always been in use, and no case of the disease had happened.

7. **The New Caledonia Epidemics.**—The terrible outbreaks among Japanese in these islands in 1892, and again in 1901, were both brought to an end by change of food. In the former case rice was entirely, in the latter almost entirely, discarded, in favour of bread.

8. **At Diego Suarez.**—During the epidemic described by Schuttelaere in 1900, which affected Senegalese and Creole soldiers, native Malagasy prisoners and Chinese labourers, all measures—even large reduction in the proportion of rice eaten—failed to check the disorder until the more radical one was taken of 'the complete suppression of rice in the native ration and the substitution for it of bread, and *pain de guerre*, and later, of *padi* . . . , and the epidemic was extinguished at once'; of which result, with the control-factor, which made it so valuable, that the Chinese, who would not have their diet altered, continued to suffer, Schuttelaere well says that it has '*toute la valeur d'une experience concluante*.'²

9. **At Dakar in Senegal.**—The epidemic described by Lasnet among prisoners fed on rice was terminated by the substitution thereof of bread. This fact, which I take from Schuttelaere³ (for I have missed Lasnet's account), formed one of the considerations which led the former officer to adopt the same successful measure at Diego Suarez.

10. Among **Indian Army Recruits** at Vizianagram, in 1897, and again in 1899, as already related, it was the substitution of, in the first case, a 'better quality,' and in the second, of 'boiled' for 'kutchá' rice, that ended the outbreaks.

¹ *Journal of the American Medical Association*, May 10, 1902, p. 1244.

² *Arch. de Méd. et de Pharm. Mil.*, December, 1901, p. 477.

³ *Loc. cit.*

11. **At Kediri and the Three Soerabaya Hospitals**, as related by Vorderman, the epidemics came at once to an end when 'stripped'—i.e., stale white—rice was replaced by 'unstripped,' generally red, rice.

The foregoing are among cases already once recounted. But the literature of the subject and daily experience offer a profusion of similar instances—epidemics, groups of cases among persons in whose circumstances the sole common condition preceding the disappearance of the disease has been change in diet.

12. **Malays Abroad.**—Such are afforded frequently in Malaya by small parties of Malays who go to the jungle collecting forest produce. After a few weeks or some months of this life, which (except for their diet) is the healthiest possible, beri-beri occurs, attacking not seldom all the party. They have been all this time on *stale* rice. They struggle home, where, if not moribund on arrival, *feeding on fresh rice*, they speedily recover.

13. **On Ships.**—The full tale of the ships whose crews have been stricken by the woeful malady will never be told. But at least many hundreds of such epidemics have been recorded, and their number seems to increase year by year. Thus, Rees¹ refers to 41 ships arriving at the Port of London with beri-beri between 1890 and 1897. The Norwegian Committee on Beri-beri² to 66 such ships touching at Norwegian ports, 1893-1898. Bullmore³ mentions 43 landing cases at the single port, Falmouth, 1896-1901.⁴ The Royal Commission on Arsenic Poisoning collected reports of 21 arriving at five different English ports between September, 1901, and October, 1902.

In some of these cases the entire crew has been attacked, so that the ship has barely made her port. The disease may have prevailed for weeks and months before the voyage ended; but it has never been recorded in any one of these numerous ship-outbreaks that the epidemic has *continued* after port was made. On the contrary, so soon as land is touched and fresh provisions have been secured, the sick begin to recover, and never another fresh case of the disease occurs.⁵

Such a continuing series of epidemics all ending in the same common event has, it seems to the writer, an evidential value

¹ *Lancet*, September 12, 1900.

² 'Indstilling,' p. xxix.

³ Report, 1901.

⁴ *British Medical Journal*, 1897, p. 747.

⁵ Nor, it is worth insisting for those who yet believe in the infectiveness of the disorder, has any new 'focus,' or any new case of beri-beri ever been anywhere set up by the coming of these beri-beri sick to land.

which fewer outbreaks, of which the conditions may have been more carefully detailed, do not possess.

But as definite instances, recorded by medical observers, in which the end of severe outbreaks was shown to be brought about by nothing else than mere change of food, the following classical cases may be cited :

14. **The 'Parmentier.'**—This unfortunate vessel set sail from Martinique on October 10, 1861, carrying 401 Indian labourers returning to their country. One hundred and forty-three only reached that destination.

Three months after departure, when doubling the Cape of Good Hope, and in mid-winter, beri-beri broke out, continuing until Mauritius was reached. There the vessel lay two or three days while water and fresh provisions were obtained. During the stay at Mauritius, and for some time after—about three weeks altogether—no more cases of beri-beri happened. But the ship had been anchored below and close to a hospital where some cholera cases were under treatment. Shortly after leaving Mauritius cholera broke out aboard, causing many deaths. Now, the beri-beri had throughout been confined exclusively to the Indian passengers ; but when cholera occurred, it attacked both Indians and Europeans, passengers, crew, and officers alike—a point for those who think beri-beri to be infectious. The cholera ceased ; but beri-beri, *of which there were no cases so long as the fresh provisions lasted*, appeared anew, and prevailed until the ship arrived (on May 10, 1862) at Pondicherry. The Indians were then 143 in number, of whom 40 were still suffering from beri-beri more or less severe, and of which 2 died that day, 5 on the morrow, and 18 more the following week.

'The few,' say Fonssagrives and Leroy de Mericourt,¹ from whom this account is taken, 'who were exempt from the disease were the cooks, two or three Indians who waited on the sailors, and got from them extras to their rations, and a few natives who had had the forethought to provide themselves before their embarkation homewards with tamarinds, chillies, preserves, dried flesh, etc.'

The immediate cessation of the epidemic on obtaining fresh food at Mauritius, and its recurrence when the fresh stuff was finished—like the similar happening invariable in scurvy—make

¹ 'Essai,' etc., *Arch. de Méd. Nav.*, September, 1861, t. xviii. See also references : Reaucar, 'Thèse,' p. 29; and Van Dieren, 'B.B. eene Rijstvergiftiging' p. 87. My quotation is from the latter.

this terrible case of the *Parmentier* an apt illustration of the point now under consideration.

15. The '*Riujo*.'—This was a Japanese naval training-ship, and her voyage, conducted under special and prearranged conditions as to diet, by way of an experimental observation, has already been referred to.

The *Riujo* left Shinagawa (Japan) on December 19, 1882, for Wellington, Valparaiso, and Callao, returning viâ Honolulu to Shinagawa, which she reached on September 15, 1883.

Within the first six weeks of the voyage there occurred 3 cases of beri-beri; in the next two months 7 cases; during the sixth and seventh months out, 125 cases. When, on July 2, the *Riujo* arrived at Honolulu, there were 123 beri-beric sick on board, and the port was only made with difficulty, 'all the stokers being down with the disease, volunteers having to be enlisted for the work. On arrival at Honolulu, the use of rice was discontinued altogether, meat and bread given *ad libitum*, and the epidemic ceased, no new case occurring on the vessel.'¹

This voyage was made at a time prior to the adoption of the reformed diet in the Japanese Navy, when rice was eaten in a very large amount, and barely supplemented by other food. This same vessel sailed again in 1887 over identically the same course as before, but using the reformed diet, and had no case of beri-beri.

Vorderman's fertile '*Onderzoek*'² offers several examples of the abolition of beri-beri from *prisons* through merely changing the sort of the rice eaten:—

16. At Japara District Prison, prior to November 17, 1895, there had been no case of beri-beri. 'On the day following this two convicts (brothers) came under treatment. Both had been incarcerated in the Preventive Prison on August 10 of that year, and on October 30 following were sentenced by the Circuit Court to death for murder.

'Owing to the occurrence of the illness, their case was under revision by the High Court. One of them died on December 3, 1895, the other on the 16th following.

'In January three other prisoners (*preventieven*) came under treatment, and in the ensuing months still more, so that when I visited the place on June 3 there had already been forty-six cases of beri-beri, eleven with fatal issue.'

¹ 'Review of the Preventive Measures against Kakké in the Imperial Navy,' Tokyo, 1890. My quotation is from Bentley, *q.v.*

² P. 120.

Everything, Vorderman goes on to say, had been done to get the epidemic under control. The cells had been disinfected, actual cases isolated in an empty portion of the old prison, extra diet was given, daily exercise of all the prisoners was attended to, the diets strictly inspected.

'Contrary to the use of natives of the Japara district, however, in the gaol itself fine scoured (*afgewerkte*) rice was given, the grains of which were almost completely freed from silver-skin (pericarp).'

In lieu of this rice, Vorderman got a sort of rice substituted, such as the outside natives were using, which was unstripped, or unscoured (*onafgewerkte*) white grain, with a considerable mixture of red grains. Also, a part of the greenstuff in the ration was replaced by potatoes.

The change was effected on June 4, 1896. A single new case of beri-beri was admitted on the 15th, and thereafter and until the date of publication of his results, not another case happened.

17. At the **Toelongs-Agoeng Prison** the experience was similar. Prior to June 30, 1895, the diet had been one of *white scoured* or stripped (*afgewerkte*) rice, and beri-beri had attacked the inmates considerably ('Kwam . . . in niet onbelangrijke mate voer'). A white unscoured rice, mixed with red, was made to replace the other, and after this not a single case more of beri-beri happened there.

Four more recent, and perhaps more important, illustrations, from institutions under the charge of British medical officers may serve to close the list of proofs of the deduction.

18. **Sarawak Prisons.**—In a discussion on the etiology and pathology of beri-beri at the annual meeting of the British Medical Association at Leicester, 1905, Dr. Charles Hose, of Sarawak, made the following among other statements to corroborate his own independent conclusion to the effect that beri-beri was the result of some poison absorbed from decomposed, or fermented, rice.¹

¹ *British Medical Journal*, October 28, 1905, p. 1099. Dr. Hose is reported as continuing: 'I would add that I have recently learned that, in consequence of this report and the representations made by Drs. Braddon and Daniels, the practice of supplying fresh rice has now been adopted in the Federated Malay States.'

The writer's views as to the dependence of beri-beri on rice have been constantly urged by him upon the Government since 1899, but until very recently without any effect. At the Congress of Medical Officers of the Federated Malay States in 1900 these views were more succinctly stated in a discussion on the subject then introduced by him. At that meeting every one of the members present

'I would refer . . . to the half-yearly report of the superintendent of police and prisons, Sarawak, dated January, 1905. In that report the results which attended the efforts of the principal medical officer (Dr. A. J. Barker) to secure a supply of fresh rice to all prisoners in that gaol are recorded. I wish to point out that in that particular prison beri-beri had been practically epidemic. For 1904 the report shows a reduction in percentage of patients amounting to 42.95 per cent. in favour of the latter year.'

disseminated from the views expressed; and since that date the strenuous efforts of most of his colleagues in the East have been directed to proving the entire futility of the 'rice-theory.' The two Directors of the Institute of Medical Research (Dr. Hamilton Wright, and after him Dr. C. Daniels), by whose opinion the Government have been guided, have both, after ample opportunities of investigation, expressed themselves in favour of the view that beri-beri is due to some form of infection. Dr. H. Wright says in his summary of 'results obtained' ('Study,' p. 61, par. 376): 'He will be a bold and badly-informed theorizer who contends that the Siam or Rangoon rice of the gaol held an active specific organism *or toxin* [my italics] after being steamed for two hours under two atmospheres of pressure. *Braddon's theory is founded on an assumption pure and simple, supported by one-sided evidence which falls to the ground* in face of the single incontrovertible fact that ninety cases of beri-beri occurred when the Siam rice used was sterile.'

Readers of this work will be by now in a position to know how little this writer apprehended what my theory really was. One may with satisfaction admit, however, that the evidence is, as Dr. Wright says, all one-sided.

Dr. Daniels' views have not yet been printed, but having had the privilege of reading in manuscript some part of a report which he proposes to publish, I am able to affirm that his opinions are at entire variance with my own. He will hardly therefore claim to have been, as Dr. Hose has suggested, a joint advocate with myself for the abolition of uncured rice—and the extinction of beri-beri thereby—from the dietary of the local gaols.

The Report of the Principal Civil Medical Officer for the Straits Settlements for 1904 has the following paragraphs:

'Prison Hospital.

'3. Dr. C. W. Daniels, Director of the Institute of Medical Research, Federated Malay States, visited the prison in February, and reported on its sanitary condition, making certain recommendations. [These were mostly building and other alterations tending to improve the ventilation, drainage, and water-supply arrangements.] Nearly everything suggested by Dr. Daniels was done—eleven additions and alterations, says Dr. Leask, the prison medical officer, who reports this—but it does not appear that among the very numerous suggestions made by Dr. Daniels was included any substitution of cured or fresh for other rice.

'5. In spite of all improvements, three cases of enteric fever occurred, two of whom must have contracted the disease in the prison; dysentery has been prevalent; one case of what was clinically cholera occurred; and *beri-beri cases have increased in numbers.*'

The report continues :

'In face of these figures it is scarcely necessary to say that the health of the prisoners has been very greatly improved, while no alteration has been made in the sanitary condition of the gaol. It is quite clear, therefore, that this radical change has been effected by the substitution of freshly-husked padi instead of the Rangoon rice used in 1903. . . . There have been 4 deaths among prisoners, as against 17 for the same period last year. . . . There has been no fresh case of beri-beri in the gaol during the half-year, the other cases—now reduced to 4 in the gaol and 1 in the hospital—being chronic.'

19. **Penang Gaol.**—The following is an excerpt from a paper read by Dr. S. H. R. Lucy, colonial surgeon, Penang, at a meeting of the local branch of the British Medical Association held there in September, 1905 :

' . . . The gaol at Penang has always enjoyed a rather peculiar immunity from beri-beri. There have not been more than seven cases treated in the gaol in any one year since 1888, and many of these are marked as having contracted the disease before admission.¹ But :—

'From April 26 to July 31 this year, no less than 38 cases were admitted to the hospital—the daily average population being 421.92. One case only—the first—was suffering from beri-beri on admission to the gaol ; 1 other had been in gaol only ten days when attacked ; of the remaining 36, it is highly probable that 34 must have contracted the disease in gaol. Twenty-six of them had been in gaol for a period of three months or more before admission to hospital.'

¹ Comment has been made upon this supposed immunity in an earlier part of this work, and the explanation suggested that the Penang Gaol gained its exemption from the fact that only short-sentence prisoners were kept there, all long-sentences being sent to Singapore. It is a coincidence which at least enforces this explanation, and also offers a reason for the novel outbreak to which Dr. Lucy presently refers, that at the end of 1904 an Ordinance came into force whereby the term of punishment included under the designation of 'short sentences' was extended from *six months and under*, which it had previously implied, to include all sentences up to *twelve months*. The result of this would be—short sentences being still kept at Penang—to provide (what there had not been before) the material necessary for an outbreak in the persons of prisoners incarcerated for a period long enough to make them, not merely suitable, but likely subjects of the disorder. It will be recollected as having been mentioned that a similar state of affairs—but then only temporary—was produced when in 1882 a number of long-sentences were transferred to Penang from Singapore. Beri-beri broke out for the first time—so it was then stated—at Penang shortly after the arrival of these prisoners.

'Of the 38 cases, 35 were Chinese, 2 were Tamils, and 1 a Malay.

'Mr. Scriven, the assistant surgeon at the gaol, where he has been for the past eight years, informs me that in April last he called the attention of the Colonial Surgeon to the bad quality of the Rangoon rice supplied to the convicts; he states that the rice about this time was worse in quality than he ever remembers being brought into gaol. In June I accompanied the principal civil medical officer upon his inspection of the gaol, and the store of rice was then condemned as being so stale and dirty as to be unfit for consumption.

'On July 25, following an order from Government, all rice-eating convicts were fed with "parboiled rice" in place of Rangoon; on July 29 the thirty-eighth case of beri-beri was admitted to hospital. From that date up to the present (September 1) there have been no further cases.'¹

20. **The Singapore Lunatic Asylum.**—To this institution, which its superintendent, Dr. W. G. Ellis, has repeatedly described as 'a pest-hole of beri-beri,' reference has more than once been made in this work.

Dr. Ellis has long been a decided and uncompromising opponent of any 'rice,' any dietetic theory of beri-beri. An incident recorded by him, together with the Jelebu episode related earlier in this work, long formed in the writer's mind an obstruction to any complete and reasonable conception of the genesis of beri-beri—above all, from rice as a source. Dr. Ellis related in the Annual Medical Report for the Straits Settlements, 1903, and again in the *British Medical Journal* (November 14, 1903, p. 1268), more or less by way of reply to the writer's views, which had then recently been urged upon the local government, a certain experiment conducted by himself thirteen years earlier, which in his view completely and for ever disposed of the possibility that beri-beri could be in any way due to rice. The experiment was that 20 patients in the asylum, who were healthy when admitted (*i.e.*, except mentally), were placed apart on admission, being located in separate cottage blocks, where they were fed on 'European food, and no rice whatever.' In less than six months

¹ Dr. Lucy in this paper sets out to urge and to endorse, on evidence recently gained, the view to which he had formerly been definitely opposed when first it was set forth by the writer. His further comments strongly support my own statements in this work. He adds: 'I have never met with a case of beri-beri in a person who was not an eater of "uncured" rice, and have never seen a case amongst Kampong Malays or Estate Tamil coolies. All these people are under normal circumstances eaters of "parboiled" or else of fresh rice.'

'more than half the cases had shown symptoms, more or less, of beri-beri.' This convinced Ellis that food had no part in the causation of the disease, and this opinion, he in 1903 reiterates, he has since seen no reason to alter. This experiment is further considered later on. The cases were probably not beri-beri.

Dr. Ellis holds the belief, which he has in frequent communications to Government insisted upon, that beri-beri is miasmatic, due to something in the soil or surroundings of the place where the disease occurs. At his suggestion Government even built a seaside institution, at which asylum patients could be treated, to the opening of which a great improvement subsequently following was attributed, not only in the recovery-rate of cases sent there, but even in the number of original cases arising in the asylum.¹ At these seaside wards *the sand beneath the buildings* was constantly

¹ Ann. Med. Rep. S. S., p. 23: 'A table at the end of this report is instructive in showing the improvement in the recovery-rate from the disease [beri-beri] after the opening of the seaside ward at Pasir Panjang in 1900, and then later *in the number of patients attacked* [present writer's italics]. . . . When advocating the seaside wards in 1899, I wrote: "My only apprehension is that possibly in time the ward and its surrounding compound may become also saturated with the germ, and so lose its powers for good." Since then two other wards have been built, the disease appears to be quite stamped out from the female side, and the few cases that occur on the male side at Sepoy Lines rapidly recover when sent to the seaside, and *do not relapse on return as formerly they so often did*. In my report for 1902 I gave some of the details of the plan of attacking the disease by thorough disinfection of all personal linen, bedding, furniture, crockery, wards, compounds, etc., and to this I imagine our success is mostly if not entirely due.'

In a memorandum previously circulated by the Government Dr. Ellis had expressed a less qualified opinion as to the benefits of the seaside treatment. Here he apparently halts between two. The seaside was certainly of a marvellous virtue if it should have prevented new cases and relapses arising in an institution miles away! This work is meant to demonstrate one theory, not to attack others; but it is impossible to avoid the reflection how much time and expense has been wasted in the futile efforts which those who believe in the infective origin of beri-beri have made to deal with its epidemics on such lines, or not to deplore the loss of priceless opportunity to demonstrate the true etiology of it which the circumstances of institutions such as the Singapore Asylum or the Singapore or Pudoah gaols would have afforded. For a single simple experiment—namely, to feed one-half of the population of any one of these institutions on cured or fresh rice or on bread for a year, while the rest of the inmates continued to use the ordinary diet of uncured rice, would have fully and finally settled the issue.

At this present time of writing the local Government have promised to give the writer facilities for conducting such a crucial test; but in the institutions most suitable for the purpose the necessary conditions are no longer present—beri-beri has as an epidemic of importance disappeared from all of them.

removed, being shifted either below low-water mark for the tide to wash it, or above high-water mark for the sun to dry it, so that the supposed beri-beri-emitting agent in the soil should be destroyed. Even these measures failed to prevent entirely the recurrence of the disease.

But in the autumn of 1904 a change was made in the rice used in the asylum; 'cured' (Bengal) was substituted for uncured rice—I do not know whether this was, as in the case of the Penang Gaol, by order of the Government—with the result that among those using the cured rice, at least, beri-beri disappeared. I quote from the Annual Medical Report of the Straits Settlements for 1905 (p. 21):

'LUNATIC ASYLUM.

'Report by Dr. W. G. Ellis, Medical Superintendent.

'6. *Beri-beri*.—From October 13, 1903, to October 13, 1904, all patients were fed on the cured Bengal rice, and during this period but one case of beri-beri occurred in the asylum—a case attacked early in November, 1903. During this same period the disease was endemic in the Singapore Gaol, and formerly, when epidemic in the one institution, it was invariably epidemic in the other. *On October 13, 1904, we returned to the uncured Siam rice, and in December 15 cases of beri-beri arose, coming from all parts of the asylum, though none occurred amongst the 20 males and 8 females kept on Bengal rice as controls*' (present writer's italics).¹

Such an experiment came near to be one of the crucial nature so long desired to be carried out in regard to beri-beri. But in this place, as one more convincing and explicit than usual, of the many proofs of the deduction which it is the purpose of this section to demonstrate, Ellis's last asylum experience seems wholly convincing—a clear instance of the termination of an epidemic upon the removal from the diet of the sort of rice which had formerly produced it.

¹ Dr. Ellis adds to this: 'I am far from being convinced that the consumption of the uncured rice is the cause of beri-beri, and have yet several experiments to complete; but my experience of the past few years, since making researches into the subject, certainly tends to make me far less antagonistic to the theory than formerly. My work is not yet ready for publication, but shortly I trust to have some results to bring forward.' It will be interesting when these results appear, in welcome addition to the labours of others in the field, to learn to whose initiative we owe it that Dr. Ellis came at last to be impelled to test once more a theory which, so recently as 1903, he claimed his own researches had positively disproved.

In no previous year since 1896 (it appears from tables given by Dr. Ellis in this same report) had there failed to occur cases of beri-beri in this asylum during the months of October, November, and December. When in 1903 cured rice was for the first time brought into use instead of uncured stuff, for the first time also cases (we except the single case, which may have been an unsuspected relapse) of beri-beri failed to appear. In that December there was no case, although at the neighbouring institute (the prison) beri-beri prevailed as usual : 53 cases happened there in November ; 65 in December. Finally, on resuming the uncured rice the next autumn, and after two months' use of it only, in December the disease again recurred.

21. In the **Singapore Prison** in 1904 was begun an observation which confirms, and in a sense supplements, the results gained in the asylum.

In the prison, beri-beri had, as has been noted, been a scourge ever since 1897. In that year, following reversion to an excessive rice-diet, instead of the one containing a very low proportion of the grain, upon which the gaol had been twelve years beri-beri-free, three cases happened in September. Each year following there were hundreds of cases.

The cases which occurred in succeeding years had generally been most numerous in the autumn of each year. The particular months of November and December together had in no single year yielded less than 24 cases (1899), December in no year less than 17 cases. In 1903 there had been but 29 cases in all the nine months (February to October), but in November that year there were 53 admissions for beri-beri, and in December 65. These figures show that, so far as the season of the year was concerned, the time at which the malady could always be relied upon to be, if not at its very worst, at least, certainly severe, were the two months of November and December. When in any year the course of the epidemic showed greater prevalence than usual in preceding months, the assured expectation would be that the last two months of the year would be even more evilly productive of cases.

In 1904 the prison had more cases of the disease than in any year preceding, except 1902. In 1902 there were 415 cases ; in 1903 there were 169 ; in 1904 no less than 266. These last were, till September, distributed fairly equally throughout the year—it is mournful that such a state of affairs should have been the only sequel to all the expensive sanitary improvements adopted

by the Government at the instance of the Director of the Institute of Medical Research—and previous records made the expectation almost certain that there would be an equal, if not a greater, access of cases during the last two months of the year.

But in November Bengal rice—*i.e.*, 'rice which has gone through the process of malting'—says Leask, the prison medical officer, 'was given, instead of Siamese rice, on November 1, except for *congee*¹-making, for which it was unsuitable.'

The result was that, instead of 118 cases of beri-beri, as happened in November and December, 1903, or even more, as the course of the epidemy (252 cases in the preceding ten months) had presaged, the total number of beri-beri admissions fell to its lowest record. Only 14 cases were admitted during the two months, 10 in November, 4 in December.

Herein it is that the prison experience supplements that of the asylum; for it was precisely in this December, 1904, when the use of Bengal (or, at least, of 'cured') rice in the prison had abolished beri-beri, or, at least, reduced it to its lowest ebb—it was in this very month that suspension in the use of Bengal rice and reversion to uncured rice led to recrudescence of the disease in the asylum.

During the first seven months of 1905 exactly the same dietary remained in force, and a certain number of cases—two or three a month—continued to occur. For this happening it is likely that the uncured rice in use for morning *kanji* (rice-gruel or porridge—'skilly') was responsible, for on August 1 cured rice was substituted at this meal also, and during the ensuing five months, but six cases in all were recorded, two being relapses.

The results in tabular form appear as follows :

Cases of Beri-beri treated at Singapore Prison during 1905.

	Entered Prison with Beri-beri.	Originated in Prison.		Total.
		Primary Cases.	Relapses.	
January to July 31 ...	22 ²	15	7	44 ³
August to December 31	nil	4	2	6 ³

¹ *Congee*, or *kanji*—rice-gruel or porridge, used at the early morning meal.

² Twenty of these were readmissions of cases previously sent to Malacca for treatment.

³ All six arose before the end of October. In November and December there were no cases.

During the five years preceding the numbers of cases treated for corresponding terms had been as under :

	1900.	1901.	1902.	1903.	1904.	1905.
January to July ...	99	111	225	42	147	24 ¹
July to December...	125	108	192	127	119	6

If there are excluded from comparison the years from 1885-1896, when one special condition—the reduction of the rice-ration to an unusually small amount, viz., 14 ounces (instead of, as at present, 22 ounces)—saved the prisoners from the disease, then it may be said that, as the result of the adoption of cured instead of uncured rice in this prison :

(1) The number of cases produced during the year became the lowest on record during its history.

(2) For the first time during thirty years, among prisoners fed on a full rice diet, the two months of November and December (in previous years invariably these most productive of the disease) passed unmarked by the occurrence of a single case.

(3) For the first time in the same long term the year passed without a single death from beri-beri.²

22. **Hose's Experiments in Borneo.**—The question of rice as in some way a cause of beri-beri having been so long in the air, it might be supposed that *direct experiments* would have been made long ago to test the connection. But, apart from those of Takaki, carried out entirely from the dietetic point of view, I know of none earlier than those of Vorderman, already referred to, and one made by Hose upon natives of Borneo, of which I quote an account :³

'In May of last year (1900) Mr. Charles Hose, D.Sc., who is the Resident in the Baram district of Sarawak, handed to Dr. Strangeways Pigg, of Cambridge, a paper in which he stated the results of investigations into the origin of beri-beri, which

¹ The readmissions mentioned above are excluded.

² For the above facts I am indebted to Dr. W. K. MacDowell, C.M.G., P.C.M.O., of the Straits Settlements, and Drs. W. G. Ellis and R. Dane, the officers lately in medical charge of the prison.

³ From the *British North Borneo Herald*, June 17, 1901, extracted from the *Morning Post*. This was my original reference. But I see (*British Medical Journal*, October 28, 1905, p. 1099) that an abstract of Hose's original paper appeared in the *Medical Review*, June, 1901.

he had been carrying on for some years in Borneo. Mr. Hose had himself been a sufferer from the disease, and it was mainly this circumstance that induced him to turn his attention to the subject.

'The conclusion at which he arrived was "that the principal cause of beri-beri in Borneo is to be traced to mouldy rice." Mr. Hose came to the conclusion that rice is instrumental in causing the disease by a process of exclusion. He found that in Borneo the disease was much more prevalent among men than among women, that it was frequently contracted, as in his own case, on a journey in the jungles of the interior; that it was more prevalent at certain periods of the year than at others, and that outbreaks were frequent in gaols and among the Chinese coolies employed on tobacco and other plantations. *The women in Borneo, who very rarely leave the villages for any length of time, live mainly on freshly-husked rice*, while the men are frequently absent in the interior on rubber-collecting expeditions for several months at a time. They carry with them their rice in bags, and after a time in the damp climate the rice becomes mouldy. Imported bazaar rice, chiefly from Java and Siam, is given to the inmates of gaols and to the coolies on the plantations; and Mr. Hose further ascertained that the outbreaks of the disease in the Baram district are most noticeable in the months of April, May, and June, immediately after the period when the north-east monsoon has made it impracticable for ships or native craft to enter the Baram River. To meet the local requirements large quantities of rice are imported before the monsoon period for the consumption of traders and others who do not cultivate their own *padi*. Probably this rice has been in bags for some weeks or months before it reaches Baram, and after being kept in bags for a further period it naturally becomes musty and mouldy.

'Microscopic examination satisfied Mr. Hose of the existence of a minute fungoid growth on rice of this description, and having, as he believed, found a possible cause for the disease, he proceeded to make further investigations.

'*Thirty-nine* male Dyaks who had contracted beri-beri during their expeditions in the jungle, where they had been living on bazaar rice, were placed in villages where none but freshly-husked rice was used. Of the thirty-nine men thirty-three recovered and six died. *A hundred and twenty-eight* Dyaks who had contracted the disease under similar conditions continued to

live, after their return to the coast, on imported rice, and of this number forty-seven died.

'The death-rate of cases fed on fresh rice was thus 18.1 per cent., as compared with 37.5 per cent. on mouldy rice.

'Having made out what he believed to be a *primâ facie* case against the mouldy rice, Mr. Hose tried the experiment of feeding monkeys (*Macacus nemestrinus*) on bazaar rice. One of the three monkeys experimented on exhibited no special symptoms, but the other two developed some of the characteristic reflex actions of beri-beri, and showed a decided lack of energy, which is one of the symptoms of the disease. But in none of the animals did the usual œdema in the lower part of the legs occur.'

23. **Experimental Determinations of Comparative Toxicity of Different Rices—Conditions Necessary to be observed.**—Prior to any knowledge of Hose's work the writer had been experimenting in the same direction in hospitals.

In attempting to determine the relative effect of two sorts of rice upon persons already having beri-beri in hospital, certain fallacies must be guarded against, which will otherwise vitiate the conclusions obtained :

(1) Since both the prevalence and case-mortality of beri-beri vary, not only from year to year, but month by month, the cases, the subject of experiment, must undergo the test simultaneously, all other circumstances being, of course, made equal.

(2) For the same reason no comparison can be made between the results obtained during one year or season and another, nor even between those obtained, though simultaneously, at two different places. Since, whatever the essential cause of beri-beri, it varies in intensity, periodically, and whatever the manner in which it is applied, the extent of application varies in different places, so that the condition of patients, and therefore the degree to which they will react to any form of treatment or diet, must be taken as unequal under these different circumstances.

(3) Finally, looking to the conclusions which may be expected as the result of an experiment carefully devised and carried out on the above lines, it may be well to remember that while positive results have always a positive value, negative have none.

In the trial of the question whether some sorts of rice have or have not a causal relation to beri-beri, this is especially so. For

it is a part of the rice theory (as postulated in this work) that the degree of noxiousness, even of a sort of rice which is usually poisonous, should vary, that at times it may be entirely free of poison.

When, after feeding two bodies of patients on different rice, no difference should be obtained, therefore, the negative issue would not prove as to either of the two rices tested that it was never noxious. The logical inferences possible would be either: (1) That both rices were *then* equally injurious; or (2) that both of them were *then* equally harmless.

Its frequent repetition might certainly lend to the negative result something more than the force of a mere presumption; but this, a single observation showing positive differences in the effect of the two rices, would be sufficient to destroy. On the other hand, the demonstration of conspicuous difference in the results, if only upon a single occasion, or in one term of observation, but affecting a considerable number of cases, would, when all other conditions were equal, be proof of real influence on the part of one or other of the two rices upon the disease—an influence only explicable on the assumption that one of them was more or less actively toxic.

24. Fry's Experiment at Penang.—Experiments carried out by Dr. W. S. Fry, the colonial surgeon resident at Penang, at the Pauper Hospital in 1902, afford some proof of the actual toxic influence of one sort of rice upon beri-beri, while they illustrate also the difficulties attending its demonstration. For permission to use these I am indebted to Dr. Fry and Dr. Kerr.

For the latest part of the results I am indebted to my friend Dr. Travers, who has recently printed and circulated them.¹

Dr. Fry's observations show that even at a season of the year (September to December) when the case-mortality observed in hospital was at its worst—397 per 1,000—it was reduced by feeding the patients on 'cured' instead of the usual uncured rice by 26 per 1,000; for the period June to December, by 28 per 1,000.

In each of five consecutive quarters during which the experimental feeding was continued, although the gross mortality varied greatly, there was a uniform difference in favour of the 'cured' rice-eaters.

¹ In a 'broadside' purporting to show the entirely negative character of the results.

In the last three months of the trial when the case-mortality, was at its worst (and unusually severe), little impression was made, but the actual difference in death-rate observed was *26 per 1,000 less in the case of the cured rice-eaters.*¹

In the four months, January to May, when over a hundred cases on each rice were on trial, and the gross mortality was at its lowest, the difference was no less than 220 per 1,000, the mortality on 'ordinary' (Siam or Rangoon uncured) rice being 265, and on cured rice only 45 per 1,000.

On the whole series of 686 cases, nearly equally divided, the difference in favour of the cured rice was at the rate of 58 per 1,000.

I append the complete series of results so far obtained in tabular form :

Result of Feeding Patients Admitted for Beri-beri at Penang Pauper Hospital on 'Cured' or 'Kling,' and 'Uncured' or ordinary, Rice respectively.

	Cases on Uncured Rice.			Cases on Cured Rice.			Total.		
	Cases.	Deaths.	Mortality per 1,000.	Cases.	Deaths.	Mortality per 1,000.	Cases.	Deaths.	Mortality per 1,000.
September to December, 1901..	104	13	125	46	5	108	150	18	180
January to May, 1902	113	30	265	109	5	45	222	35	157
June to August, 1902	21	7	333	21	6	285	42	13	309
September to December, 1902..	129	53	410	143	55	384	272	108	397
Totals and means	367	103	280	319	71	222	686	174	253

Assuming that the circumstances of these patients were made as equal as possible, and that the cases were throughout assigned to one or other of the divisions, as admitted, without selection—and Dr. Fry states that this was the case—the conclusion from the experiment must be that uncured rice is prejudicial to beri-beri patients, and that some saving of life—at times a very great saving—may be effected by eliminating it from their dietary.²

In these returns no distinction is made of deaths happening at

¹ Dr. Travers, in his 'broadside,' calls this 'practically no difference at all.'

² The experiments were continued : '7. The Kling rice treatment, which was begun in September, 1901, by Dr. W. H. Fry, is still being continued for beri-beri cases in the same proportion as last year, half of the admissions being

particular periods, and it may be objected that the inclusion in them of cases brought in moribund, or dying shortly after admission, will prejudice the results. But it was a condition of the experiment that *no selection* should be made of cases, which were placed alternately on one diet or the other in the order in which they came in. The number of patients in a condition which no change of diet could affect would thus, according to ordinary chances, be equal in either group.

In the writer's view every case of beri-beri—leaving out those actually moribund—must be influenced by the continuance or discontinuance of the poison which produced the disease. Every additional meal of rice (if in it lie the cause) will exert its effect, will tell the more, the more desperate the condition of the patient is.

But to meet any possible exception which may be taken to the conclusions on this score, in the writer's own series of cases, those dying before ten days in hospital are classed separately, and the mortality is reckoned separately for them and for the survivors. The effect of change of rice is exhibited in both groups.

25. The Writer's Experiments on Cured and Uncured Rice in Hospitals.—In Negri Sembilan during 1901 and 1902 the conditions for making observations were not favourable, the prevailing case-mortality of beri-beri in the hospitals being low—79 and 60 per 1,000 respectively.

With such a rate, and when deaths due to complications are excluded, it will be seen that not much margin is afforded for testing the effect of any two factors upon the death-rate.

Nevertheless, results were obtained proving that uncured rice does definitely increase the mortality from beri-beri, or—what amounts to the same thing—the use of cured rice reduces it.

In the first quarter of 1901 *all* the cases had uncured Rangoon rice, and the case-mortality was as follows :

<i>Within ten days</i>	62 per 1,000
<i>After ten days</i>	44 per 1,000
242 cases were admitted.				

During the remaining three quarters of the year all patients had 'cured' ('Penang parboiled,' or 'Kling') rice.

placed on Kling and the other half on Chinese rice. There were 234 admissions for beri-beri with 69 deaths, giving a percentage of 24·40 on Kling diets and 25·67 on Chinese diets. Of the above deaths ten died within twenty-four hours of admission.'—Dr. T. C. Mugliston in Annual Medical Report of Straits Settlements for 1903, p. 40.

VARIES WITH VARIANCE IN RICE-SUPPLY 313

The rates were :

<i>Within ten days</i>	48 per 1,000
<i>After ten days</i>	25 per 1,000

827 cases were admitted.

In 1902 each alternate case admitted was placed on the one or the other of the two sorts of rice and kept upon it until discharged.

This was done at each of five hospitals.

At each of them a difference in mortality followed in favour of cured rice.

Including every death, the rates of mortality were :

On the <i>uncured</i> rice	89 per 1,000
On the <i>cured</i> rice	66 per 1,000

Excluding certain deaths due to intercurrent complaints, the mortality *before ten days* was reduced from 61 to 32, and *after ten days* from 29 to 22 per 1,000 by the uncured rice.

The details appear below :

Table showing the Results of Feeding Beri-beries on Cured and Uncured Rice in Negri Sembilan, 1902.

Uncured Rice.	Admissions.	Deaths.		Case-mortality per 1,000.	
		Before Ten Days.	After Ten Days.	Of all Admissions within Ten Days.	Of Survivors after Ten Days.
Seremban	175	13 ^a	8	74	49
Port Dickson ..	99	—	—	—	—
Tampin	18	4	1	222	71
Kuala Pilah ..	33	3	—	91	—
Total	325	20	9	61	29
Cured Rice.					
Seremban	177	8 ^b	7	45	41
Port Dickson ..	93	—	(2) ^c	—	(21)
Tampin	18	3	—	166	—
Kuala Pilah ..	43	2	—	46	—
Jekebu	92	3 ^d	2 ^e	32	22
Total	423	16	9	37	22

^a, Nine within forty-eight hours; ^b, six within forty-eight hours; ^c, one of acute dysentery at thirty days and one of acute diarrhoea at forty-three days after admission; ^d, all within forty-eight hours; ^e, a third death occurred from remittent fever—the other of the two had cirrhosis of liver also.

314 THE CAUSE AND PREVENTION OF BERI-BERI

Adding together the results obtained at Penang and in the five Negri Sembilan hospitals, we have :

	Fed on Uncured Rice.			Fed on Cured Rice.		
	Cases.	All Deaths.	Case-mortality.	Cases.	All Deaths.	Case-mortality.
At Penang	367	103	280	319	71	222
At Negri Sembilan..	325	29	89	423	25	58
Total	692	132	369	742	96	280

Total cases observed, 1,374.

The reduction in mortality accompanying the use of the cured rice was thus at the rate of 89 per 1,000.

The considerable number of cases dealt with (1,374 are tabulated), and the constancy of the difference observed in favour of the cured rice at each of the six hospitals throughout the period of fifteen months covered by the experiment, and the mode of experiment itself preclude the explanation of these results by chance. They must be taken as so far furnishing an additional and direct proof therefore of the dependence of beri-beri upon uncured rice.

As a final instance, and embodying the latest acquired and most striking evidence of the prejudicial effect of a continued diet of uncured rice on those already stricken with beri-beri, may be submitted the following experiences gained at Christmas Island.

This small dependency of the Straits Settlements was in 1891 inhabited by but nine persons in all. In 1900 coolies were introduced by a company engaged in removing deposits of phosphates found there, and down to date it has had a considerable population of mixed races, chiefly Chinese.

Beri-beri, breaking out shortly after their arrival, soon assumed the proportions of a terrible and destroying epidemic among these labourers, almost all of them becoming affected, and many—in one year one-third of their whole strength—dying of it. In 1903 the island became the scene of operations of a special Commission sent out under the auspices of the London School of Tropical Medicine to study the disorder. The fruit of those labours (unfortunately inconclusive) is embodied in a paper contributed by H. E. Durham, the leader of the expedition, to the *Journal of Hygiene*, January, 1904, some further references to which are made elsewhere in this work.

VARIES WITH VARIANCE IN RICE-SUPPLY 315

The following table presents information kindly furnished me by the present medical officer of the island, Dr. MacDougall, through the district officer, my friend Mr. W. T. Chapman :

Some Statistics of Disease at Christmas Island, 1901-1905.

Nationality.	Year.	Average Strength of Population.	Beri-beri.		Other Diseases.	
			Cases.	Deaths.	Cases.	Deaths.
Chinese ..	1901	660	670	226	619	46
	1902	656	673	88	804	85
	1903	732	587	59	820	14
	1904	785	971	92	865	13
	1905	944	489	11	1,336	9
Malays ..	1901	20	2	2	9	3
	1902	20	2	—	4	—
	1903	30	3	1	2	—
	1904	38	2	—	4	—
	1905	45	2	—	6	—
Tamils ..	1901					
	Jan. to Nov.	10				
	Nov. to Dec.	40 ¹	15	1	12	—
	1902					
	Jan. to March	40 ¹				
	March to Dec.	8	17	3	19	—
	1903	7	—	—	—	—
Bengalis ..	1904	6	—	—	2	—
	1905	4	—	—	—	—
	1901	18	—	—	1	—
	1902	18	—	—	3	—
	1903	24	1	—	4	1
Europeans ..	1904	26	—	—	1	—
	1905	34	—	—	4	—
	1901	12	—	—	—	—
	1902	12	—	—	1	1
	1903	14	—	—	—	—
	1904	13	—	—	—	—
	1905	15	—	—	—	—

N.B.—The 1905 returns up to November 30 only.

Never less than 50 per cent., sometimes more than 100 per cent., of the Chinese coolies employed were admitted to hospital

¹ In November a party of thirty-six Tamils arrived, and stayed till March, 1902. About half these individuals took beri-beri.

for beri-beri annually! In the worst year a mortality of 342, in the best, of 12 per 1,000 living, from this single ailment.

In addition to the example it affords of the appalling possibilities of beri-beri as an epidemic malady, the table emphasizes instructively several of the deductions which have already received illustration in this work.

The limitation of the disease to rice-eaters was marked. The three races—Chinese, Malays, Tamils—who fed on rice suffered; the two—Europeans and Bengalis—who did not use rice were, with a solitary exception, continuously exempt.

The inclusion on this island of Tamils among those severely affected, the race so completely protected by their cured-rice-eating habit from the disease in Malaya, lends great force to that explanation of the latter's immunity. For on this island all the Tamils were, like the Chinese and all the labourers, fed on uncured (Siam) rice.

Again, the unusually severe incidence on those who chiefly suffered, the Chinese, enforces the truth of the deduction that, other things being equal, the severity of the malady is proportionate to the quantity of rice eaten. For at Christmas Island, as a conspicuous difference from all other places where Chinese are employed, the ration of rice allowed was not merely liberal or excessive—it was *unlimited*! The custom here was—so Mr. Chapman, the district officer, informs me—for rice in quantity to be placed in tubs in the eating-sheds, whence the coolies were permitted to take as much as they chose!

In hospital, cases were equally allowed rice *ad libitum*. Under these circumstances, the admissions to hospital for beri-beri were, as is seen, more than cent. per cent. of the actual population (many cases being more than once attacked), and, what is no more surprising, one in every three cases admitted to hospital died there. Such was the state of affairs in 1901.

The medical officer placed in charge of the island in 1902, Dr. Giddy, decided to limit the amount of rice taken by hospital patients, and there followed after this partial reduction of the rice component considerable diminution in the mortality from beri-beri, as the table shows—1901, mortality 342 per 1,000; 1902, 134 per 1,000; 1903, 80½ per 1,000.

Dr. MacDougall took charge in April, 1904. Arriving from a country (the West Coast of Africa) which had afforded him no experience of beri-beri, his views of its etiology were those of current and accepted authority. Such leaders as Manson and Scheube had then, as now, pronounced in explicit terms that the disease, whatever else it depended upon, was in no way caused by food.

The Chinese patient, whatever his condition, clamours at all times to be filled with rice, and Dr. MacDougall could see no reason why they should not have as much as they wished of this bland and easily digested nourishment, so suitable for all invalids.

Here I may venture to quote directly from my correspondent: ¹

'After Dr. MacDougall took over from Dr. Giddy,' says Mr. Chapman, 'he, going on what he had taken from the text-books, put the patients on full rice diet, with the result that the death-rate went up very considerably—to such an extent, in fact, that he was almost in despair.

'I came down here for the first time in October, and arrived on the 28th or 29th. When I found how bad beri-beri was—it happened then to be a very bad time—I told Dr. MacDougall of your treatment, and the result from it. This apparently decided him to make a change. From *November 4, 1904*, he took away entirely the rice from beri-beri patients, and the result was most marvellous and immediate.

'There was a beri-beri death on the 5th, another on the 6th, also on the 7th and 15th, and after that not another until January 18. You will see from the two tables I enclose what the further results have been.'

The Table shows the distribution of deaths from beri-beri in the island during the years 1904 and 1905.

Deaths from Beri-beri at Christmas Island, 1904 and 1905.									
<i>All Cases on Rice.</i>					<i>All Cases on Rice-free Diet.</i>				
1904.					1905.				
January	7	January	1
February	10	February	1
March	2	March	2
April	11	April	—
May	11	May	1
June	4	June	—
July	9	July	—
August	12	August	1
September	8	September	1
October	14	October	2
November ²	7	November	2
December	—	December	1
Total deaths	95	Total deaths	12
971 cases admitted.					489 cases admitted.				

¹ Apologizing to Dr. MacDougall, whose account at first hand I have no opportunity of obtaining before this book issues from the press. The facts are, however, as given directly to Mr. Chapman by Dr. MacDougall, and are published by his permission.

² Rice was abolished from the diets on November 4.

The gross mortality of the series was thus reduced from a rate of 98 per 1,000 on the rice-diet to 24 per 1,000 on the diet free of rice. Of the total deaths which did occur after the rice was taken away, which numbered 19 in all, 4 happened within five days of the change, too soon, no doubt, for the good effects of its deprivation to become manifest. Three other deaths were complicated—one by cerebral hæmorrhage, one by gastric ulcer, and the third by acute miliary tuberculosis. Deducting the latter three from the mortality properly attributable to beri-beri during the year 1905, we have the case-mortality for that year correspondingly lowered—to 12 per 1000.

I have earlier in this work commented upon the extreme variability of the death-rate in beri-beri, and have instanced a series of 100 consecutive cases treated without a death. Dr. Macdougall states also that the type of case treated during 1905 had been much less severe than formerly, most of them being of the kind which Scheube designated as 'rudimentary.' In even rudimentary cases, however, a fatal issue, as the practitioner knows but too well, is not uncommon. Thus, making allowance for all the reasons which may be held to discount the value of Dr. MacDougall's measure in depriving his patients entirely of rice, it seems to the writer impossible to doubt that the reduction in mortality among them was largely and directly due to it—that this treatment, in fact, must have saved absolutely many lives. Conversely, the truth so demonstrated makes it obvious that to continue to feed a beri-beri patient on rice—at all events, upon uncured stale rice—is to prejudice his chances of recovery.

Clinicians have long before to-day recognised the evil effect of rice on beri-beri patients. Simmons,¹ for instance, who says that his investigations (in Japan) 'justify the conclusion that it [the exciting cause of beri-beri] is a specific miasm, or ground exhalation,' yet also states that 'rice, even of excellent quality, is badly tolerated by the stomach of the beri-beri patient, is often rejected, and appears to aggravate the symptoms.'

The writer has, in the comparison of the effects of cured and uncured rice on patients made in his hospitals, shown the greatly prejudicial effect of uncured rice. Dr. MacDougall's results not only emphasize this belief, but they suggest (what had not impressed itself upon him before) that *all, even cured, rice is injurious to the beri-beric*, since the mortality among his cases which had no rice at all was so much less than among my cured-

¹ Duane Simmons, 'Med. Rep. Imp. Marit. Cust.,' March 31, 1880.

rice-fed cases. This result, entirely conforming with the theory, should, indeed, have been deduced from it, since, as will appear in a later section, the probability is that the toxic agent is not found only in stale uncured rice, but is present, though in too small a degree to produce symptoms, both in fresh and cured rice also. The difference in effect of the differently dressed sorts of grain is probably one less of quality than quantity. The toxicity of the stale stuff is due to the *increase* in it of a poison, already present to some extent in raw padi. Further growth of this is prevented by the curing process, but such poison as has been already formed in the grains is not thereby destroyed.

Even cured, and perhaps fresh rice, therefore, may contain some amount of the beri-beri poison, and every, even the slightest, addition of such poison to his diet must be conceived to be injurious to the patient already suffering from its effects.

The practical lesson emerges from those considerations that rice in any form should be forbidden to those suffering from beri-beri.

It is obvious that the causation of beri-beri through uncured rice might be, not, indeed, more clearly, but more completely shown, more simply and precisely demonstrated by the test of applying these conclusions to *prevent* its occurrence among persons otherwise liable to get it.

Could a sufficient number of individuals be restricted to the use only of cured rice for a year and compared with an equal number fed, for the same period, upon uncured rice, at some place at which beri-beri was known to be prevalent, the event (provided beri-beri did occur among any of them) would afford evidence for the theory, which would convince those who are unable to appreciate the cogency of the wider arguments already considered. A gaol or asylum in which beri-beri was prevalent would offer exactly the conditions required. But the author has hitherto been unable to obtain the use of one of these institutions for the purpose.

Proofs of Rice Theory recapitulated.—To recapitulate : Among the principal deductions from the rice theory it has been, so far, proved :

(1) *That in places where beri-beri is endemic and in communities in which it prevails, only those who eat rice are attacked ; those who eat no rice escape.*

(2) *Among rice-eaters in endemic areas, or during epidemics,*

those only who eat certain—'uncured' and stale—sorts of rice suffer ; those who eat only other—'cured' or fresh—sorts escape.

(3) *In epidemics, as with persons, where such uncured rice is consumed, the extent (prevalence and severity) of the disease varies directly with the quantity eaten, absolutely and relatively.*

(4) *The disease persists when the sort of rice eaten when it was acquired, continues to be used, and disappears when this is changed.*

The truth of these deductions has been established by the evidence of abundant facts, as to the accuracy and authenticity of which there can be no question.

The inferences themselves are such as could be made from no other theory of the disease which has yet been advanced.¹

Upon such grounds any proposition may be claimed to have been logically established, and this position must be conceded to the rice theory of beri-beri as it has been formulated in this work.

The validity of a conclusion thus completely proved will not be affected by instances apparently conflicting with it, of which the true relation to their cause may not be immediately seen. But (it will be shown later) there are no observations recorded of which complete explanations may not be found, in one or another of the many conditions which the experiences detailed indicate to be capable of promoting or preventing the disease.

Beri-beri in the East and the tropics is thus a *grain-intoxication*, of which rice, the staple grain, is naturally the common source of derivation.

The beri-beri of the West and of the Ocean—of the ship epidemics—therefore, if it is to be regarded as the same disorder, must own a similar origin—must be a grain intoxication, too.

In many of the epidemics of asserted beri-beri in temperate climates no connection with rice has been traced. It is true that all the possible channels of access of rice or rice-derived poisons have seldom been investigated. There are other ways of consuming it than the direct eating of rice as food, which will be dealt with later. But it is possible that, even as might naturally be expected, there are some instances of beri-beri with the production of which rice is in no wise concerned.

The occurrence of such cases, even epidemics of them, however, impairs neither the completeness of the proof that by far the most common source of beri-beri is rice, nor the unity of the conception

¹ Or, one may add, which seems possible. Of the remaining deductions, there is, as has been observed, none which those acquainted with beri-beri will not recognise as agreeing with experience.

of the disease, as being due to the operation always of the same poison, although derived from various natural sources or conveyed through different media.

But what cereal may be the vehicle of beri-beri in temperate latitudes can at present only be guessed, since investigation has never been carried in this direction. Some speculations on this question, and indications of ways in which rice may become a source of beri-beri *indirectly* appear later.

* * * * *

In the light of the proof given that it is upon a toxic agent occurring commonly in rice that beri-beri depends, the explanation of many extraordinary and conflicting features of the disease becomes simple.

It is thus obvious how, although neither contagious nor infectious, and not multiplied therefore by personal intercourse, it is nevertheless transportable, and its effects seem to be 'spread' by traffic. The introduction of the disease by immigrants into a country previously free from it, and its subsequent extension to the indigenous inhabitants (as Hagen¹ states occurred in New Caledonia) becomes an instance no longer of the acquisition of the disease by the latter in a manner suggesting infection, but, rather, of depravation of food habits—the adoption, for cheapness and through force of example, of an article of diet unfortunately often poisonous.

The many more, and more extraordinary instances of persons who, themselves free of the disease, and coming from a region free of beri-beri, have yet acquired it in a country where it had never appeared before, where it did not spread from them to the other inhabitants with whom they mixed, and whence it entirely disappeared with their own departure, become now for the first time intelligible, in the knowledge that the disease is conveyed in food. Such was the case with the Annamese in New Caledonia, the Indians in the Soudan, the coolies leaving India for Martinique, the Africans going back to Sierra Leone from Panama. In all such instances the victims, like the Malays on their jungle excursions, certainly carry the cause of the disease about with them, but that cause is not an infection—the 'death is in the pot.'²

¹ *Rev. Méd. de l'Est.*, t. xxv., January 15, 1893, p. 42.

² 'Forty cases of beri-beri with three deaths occurred at the St. Louis Exposition (1903) among the Filipinos present at the Exposition (*Gaz. degli Ospedali*, Milan, vol. xxv., No. 145).—*Journal of Tropical Medicine*, September 1, 1905, p. 270.

All over the world, wherever rice has been carried, and wherever it has been adopted by the people as a staple of diet, the effect has been the same : beri-beri has followed in its wake.

This has been no less evident in those countries in which the food of the people has always been rice than in those where, as an attractive and cheaper article of diet, it has newly displaced the wonted staples of manioc, or yams, or maize. For in the former, the rice originally eaten being 'home-grown,' has also been innocuous because used '*fresh*'; while, when the demands of growing populations have rendered this the local product insufficient, that which has been imported to supplement it has been of the 'white, uncured, stale' variety, the use of which beri-beri has been seen everywhere to follow.

Of the latter state of affairs Japan, British Malaya, and the Dutch Indies present examples ; of the former, South America, Africa, and Australia.

How the use of rice, even in excess under the belief that it is an efficient substitute for fresh vegetables, has come to be common with squatters in Australia, leading to beri-beri there, has already been mentioned.

The increasing spread of the same habit in South America will be referred to later on. But it is only recently that identical conditions attending the opening up of South Africa have entailed the production of beri-beri among both natives and Europeans there.

Dr. W. Darley Hartley, editor of the *South African Medical Record*, writing to the author in regard to the epidemic among Boer prisoners at St. Helena, described by Mosse, Casey, and others as beri-beri, says of the disorder there seen that it 'partakes of the features both of beri-beri and scurvy. Some men set it down as an aberrant form of both ; others look upon it as a distinct entity. We continually have epidemics of it, and almost always among prisoners in gaols, or natives in compounds (mines). But from the scurvy point of view it is curious that it yields in no way to dietetic treatment, and, further, the diet in the places affected is good enough. But there is this to be said, that in the gaols, although not in the mine compounds, *rice* enters rather largely into the diet, and it may be of bad quality. And, again, whenever it has broken out amongst people not in institutions, it has been invariably in the very dry north-western parts of the Cape Colony, and the contiguous Griqualand West and Bechuanaland, where the constant dietetic feature is plenty

of meat and an almost entire absence of vegetables. *Now everywhere in South Africa rice is used as a substitute for vegetables when the latter are not obtainable* [present writer's italics]. 'The cases we see,' adds Dr. Hartley, 'almost always assume the dry form, and they are not fatal nearly so often as the disease in other countries.'

Again, it is a result of the various conditions which have been shown to influence the incidence of beri-beri—viz., the duration of consumption, the sort and the quantity eaten, all of which are in turn affected by other and purely economic factors, such as the price of commodities, abundance of local harvest, etc.—that at different times and places, unusual and capricious, divergent, and even apparently opposed, features should characterize the malady.

Of what other disease can it be affirmed, or how in the case of a 'miasm' or an infection should it be intelligible, that its victims should be *selected*, now according to *religion*, now according to *wealth*,¹ or even (as in many cited instances) according to *race*, although no race is known to be exempt? That among people at large *men* should so often, women so seldom, be attacked, although in gaols, asylums, and homes the sexes show no difference in susceptibility? That among males the *younger, healthier*, and more vigorous especially should suffer,² although in enfeebled states the disease finds special opportunity and strikes hard? That the *unmarried* should suffer more than the married? That there should be distinctions in its incidence upon the insane, among whom *epileptics* are most prone to succumb?

Commonest of all the seeming inconsistencies of beri-beri is the fact, everywhere noted by reliable observers, that the settled inhabitants of the 'endemic' area, whether in Java or Sumatra, Borneo or Japan, Burmah or Madagascar, always remains immune from the malady, however fiercely it may 'rage' among newcomers. Strangers of every race who dwell beside them become its victims; and they themselves even succumb to it in places where they also are strangers—i.e., in any other 'endemic' area other than that in which they are at home.

¹ 'In Japan,' Baelz says, 'people in comfortable circumstances are attacked actually oftener than the working classes and the proletariat. Scheube is of the same opinion.'—HIRSCH.

² 'The experience of the East Indies, Japan, and Brazil is, that people of strong physique take beri-beri much more often than the weakly. . . . It is only the previous attacks of exhausting diseases, such as dysentery and protracted ague, that appears to increase the predisposition.'—HIRSCH.

This, which may be claimed to be a feature, as well-authenticated as it is widespread, of beri-beri, robs the word 'endemic' of any value as applied to countries in which it usually occurs. For the proper signification of 'endemic' undoubtedly is that the malady which it qualifies should attack specially and continually the people native to the area of endemicity. But in beri-beri this does not happen. It is the strangers who suffer, and who, moreover, bring the cause of the disease with them. Beri-beri is certainly nowhere endemic in the sense that it is determined by causes peculiar to, and affecting only, inhabitants of given places.

The difficult conception of an acquired immunity—an 'acclimatization'—specific only for each restricted locality, has been invoked by some to explain this phenomenon, which to believers in the infectiousness, or the miasmatic origin of beri-beri, was naturally a very extraordinary one. This absurdity—which is exactly as if it should be claimed that a Frenchman could not, while in France, acquire a disease, to which, however, Germans and others would be liable; but could do so in Germany, while the German residents should be exempt—is dissipated in the light of the truer knowledge of the disease now presented. The phenomenon is seen to be no longer extraordinary, but a simple and natural result of the conditions upon which the disease depends.

The stranger and new-comer in the 'endemic' area, it may be said, will enjoy the same immunity from beri-beri as the settled inhabitants, provided that he copies exactly in all points, or at least in the essential matter of diet, the customs of the inhabitants. The same rule applies to the native resident when he migrates elsewhere. To repeat the quaint expression of old writ, the victims of beri-beri pay their tribute to this appalling malady, 'for they know not the manner of the God of the land.'

Yet all these paradoxes and apparent caprices distinguish beri-beri, and, inexplicable upon any other hypothesis, are seen at once to be simple and natural results of the application of a poison conveyed in food. For where the wealthy are most attacked, as in Brazil (Azevedo), Féris,¹ or Japan (Baelz, Scheube), it is because they can best afford more of the rice, the relative dearthness of which (as compared with barley, corn, yams, or taro) renders it less available as a staple, by the poor, than the latter

¹ Quoted by Féris, who also states: 'All classes are attacked . . . the persons most attacked are those of the higher rather than the lower classes.'—*Arch. de Méd. Nav.*, tom. xxxvii., p. 4.

articles. But when the poor suffer most, as again in Japan (in the summer), it is because the quantity of rice necessary to produce effects, then within command of their means, is eaten undiluted by adjuncts, such as fish, which at that season become (Saneyoshi says) too expensive.

Members of one religion only suffer in communities otherwise exempt, although all share the same rice alike, merely because their creed does not permit them the dilution of the cereal with the meat or adjuncts which others use. The strong and healthy in all epidemics suffer rather than the weakly, the younger than the aged, adults rather than children, men more than women, bachelors rather than the married, as being those whom circumstances, whether of bodily vigour or domestic relationship, alone enable to eat the quantity required, in the proportion necessary to produce the disease.

The stranger dwelling in endemic areas becomes affected, although the indigenous inhabitants do not, because the latter in their own homes subsist on fresh rice (which is innocuous) instead of the toxic stale white material, forming the inevitable sustenance of those who fare forth from their own homes. The capricious local immunity of each native against a poison generated only in his own locality, suggested by Laoh and others, is thus simply explained away.

Upon such seeming vagaries in the spread or distribution of the disease most of the objections to rice as its source have been founded.

Some of these have already been dealt with. Most, it will by now have been perceived, are susceptible of explanation through the operation of one or more of the factors already referred to.

There remain other conditions, certain periodic movements in the disease, certain facts as to the production of poison in rice, derived from experimental observations upon animals, in which lies the explanation of other of the ambiguities of beri-beri.

These, therefore, will next be discussed, after which the stated objections against rice will be considered, and, it may confidently be said, disposed of.

SECTION V

PERIODIC MOVEMENTS OF BERI-BERI, SEASONAL AND MULTI-ANNUAL

SEASONAL VARIATIONS.

Beri-beri not Dependent on Climate.—A disease which can flourish equally in the hot and humid Malayan regions on the Equator, in the frozen Kurile Islands, in the dry and scorched Soudan, and at ocean in almost every latitude, obviously in no way *depends* on meteorological factors for its causation. Yet, whether for the reason that the development of active *noxæ* is favoured by one climate more than another, or that subjects are rendered by such changes more sensitive to their operation, most epidemic disorders show seasonal variations. Similarly in the case of beri-beri, it would seem likely that extremes of temperature in either direction should, by exerting depressing influence, render persons more liable to its onset. Thus, inhabitants of the tropics would succumb more readily under cold, those of temperate climates in tropical heats.

Not only so, but in a country in which a disease is endemic it is difficult to suppose that its prevalence should be wholly unaffected by meteorological changes. The agent, if it be ectanthropic, whether animal or vegetable, must, it would seem, be more favoured in development, or, at least, in the chances of its application, at one season or in one kind of weather than another, provided, that is, that it is itself indigenous to the country. If, on the other hand, although the *disease* is endemic in a country, the cause of it is something not produced, or, at least, not chiefly produced locally, but is imported, then little agreement between tropical climatic changes and variations of incidence of the disease is to be expected.

It therefore becomes a matter of some importance to determine

What, if any, relationship can be found between these two conditions.

The only one of the meteorological factors which is subject to much variation in the tropics is the rainfall.

There is little difference in the mean temperature of successive months, and those which are actually colder are not necessarily those in which most cold is *felt*. It is rather the months of greatest rainfall in which depression from cold is most likely to be experienced.

In British Malaya the rain is commonly greater at two seasons—about April and October, and generally most at the latter—but the maximum fall may happen in any month of the year. It is difficult, therefore, to trace the effect of the rainfall month by month on beri-beri, and conflicting opinions have been recorded.

Ellis,¹ Gerrard,² Travers,³ have stated that there is no relationship between the rain and prevalence of the disorder, while Wright⁴ has sought to show that there is a distinct seasonal prevalence dependent upon the monsoons.⁵

Extraordinary Factors in Periodicity.—Even when, however, distinct seasonal variation in beri-beri prevalence is demonstrable, it does not necessarily bear relation to any, however closely accompanying, local climatic changes. There are two other conditions which may, and indeed most likely do, produce seasonal fluctuations in beri-beri. Both involve the factor, on which stress was laid earlier, that the period necessary to get beri-beri is very long.

Relation to Harvest.—One of them is the fact that rice itself is an annual crop. New grain comes into the market early in each year, and is at once brought into consumption. Since it takes six months or more to acquire beri-beri, it is only when a given crop has been in use for some time that disease produced by it becomes apparent—*i.e.*, beri-beri dependent on the crop of a given year appears usually about September of the following year.

¹ 'Annual Medical Report Straits Settlements,' 1899, p. 14.

² *Lancet*, February 11, 1899, p. 367.

³ 'Annual Medical Report, Selangor,' 1902.

⁴ 'Studies from Institute of Medical Research,' vol. ii., No. 1, etc.

⁵ None of these observers have, however, offered statistics which could prove their conclusions, since with the rainfall they compare the *absolute number* of admissions for beri-beri at different times, whereas what should be shown is the *relative proportion* of beri-berics at such times to all other patients.

Immigration.—A second factor is that the *material* which chiefly yields the cases of beri-beri—namely, the newly-imported sinkhehs—also come in for the most part annually, at the beginning of each year. Were the crop and its qualities a fixed quantity, this fact, which means that the usual period of exposure to the influence of beri-beri (whether it were rice or anything else) is only accomplished for such coolies about the autumn, would alone make an apparent periodicity of the disease at that season. The conjunction of both these factors accentuates their common influence.

Age of Rice in Relation to Beri-beri.—There is a third factor, more potent, perhaps, than either of the foregoing in determining the development of beri-beri, dependent partly upon the first of them, and also upon a host of minor conditions, which operates to produce what are apparently seasonal variations in the outbreaks of the malady. This is the *age*—involving altered condition—*of the rice itself* when it comes into consumption.

By our theory it is obvious that the *noxa* in the grain being conceived to be something living, able to spread and multiply in the stripped rice-seeds, its medium—the poison, its product—must, where attendant circumstances are suitable, increase with time. The older the rice, the more toxic it will be. One need not dwell on the probable conditions which will favour or retard such change. Damp, darkness, damage of any kind, disintegration of the seeds, will undoubtedly be among them.

But when, or wherever, for any kind of economic reason it should happen that a community should be able to obtain rice so damaged only at special seasons of the year, that season, were it rainy or were it dry, would certainly be the one at which beri-beri would be most apt to occur in that community.

Of such a state of affairs there are clear examples.

Reference has been made to H. Wright's view that beri-beri happens (in British Malaya) chiefly during the north-east (November to March) monsoon. He says :

'My experience of beri-beri in the gaol and other public institutions, where the incipient signs of the disease may at once be observed, leads me to state that only a small percentage of cases contract the disease during the south-west monsoon' ('Study,' p. 7, par. 60).

And again :

'62. This is found to be true of the mines, as well as of institutions. In the Pahang Corporation Mine at Sungei Lembing (east

coast of the Peninsula) the cases of beri-beri appeared as follows during 1901 and 1902 : April, 77 ; May, 104 ; June, 32 ; July, 36 ; August, 42 ; September, 46 ; October, 43 ; November, 69 ; December, 130 ; January, 103 ; February, 135 ; March, 91.'

A statement of this kind, to have afforded grounds for a conclusion of any approach to soundness, should, of course, have included figures showing the strength of the force among which the cases occurred. But setting this aside, and accepting a real greater prevalence of the disease at Sungei Lembing at the season named, there is another very simple reason to be assigned for the happening, as the following extract from the paper by Lucy already referred to will show. After referring to the late general decrease in the virulence of beri-beri everywhere noted, Dr. Lucy says :

'At Sungei Lembing in Pahang extensive mining operations, employing some 1,200 Chinese coolies, have been going on for about fifteen years, but at this place the disease shows no signs of abating ; it is rather increasing in severity, and during the months of December, January, February, and March of each year it assumes epidemic form with a heavy mortality.

H. Wright, in his 'Study,' No. 1, entitled 'An Inquiry into the Ætiology and Pathology of Beri-beri,' refers to the heavy case-incidence of beri-beri at these mines during the months of the north-east monsoon, and says (p. 8, par. 63) : 'Just what determines this result does not appear to be clear.' I would submit that the following facts may supply the explanation :

'Sungei Lembing is situated some forty miles up the Kuantan River, which empties itself into the China Sea, on the east coast of the Peninsula.

'There are no roads or railways from the coast to the mines ; the only means of transport is by river. During the south-west monsoon the journey from the coast to the mines can be done in about two days, but through November, December, and January the difficulties of transport are much increased by floods in the river—the journey may occupy seven days or more. The transport of supplies is further rendered more tedious and uncertain by the operation of the gales of the north-east monsoon on the coast. Steamers from Singapore (which is the centre of supplies) to Kuala Kuantan are few and far between, and these are not unfrequently delayed by the heavy weather that prevails during this season ; moreover, during these months freights are high. For these reasons the management of the mines takes the pre-

caution of laying in a large stock of rice before the monsoon commences. This rice is stored at the mines, and it constitutes the only available supply for consumption by the coolies. I have seen samples of this rice at the mines, and it is not good quality.

'In January last when at Singapore the agent for the corporation mines showed me samples of Rangoon rice as supplied to the Sungei Lembing coolies. The rice was stinking, mouldy, and infested with insects. It is inconceivable that rice of that quality could be consumed with impunity. The agent told me that the particular time, about October, at which he had to send large consignments of rice to the mines coincided with an interval between the crops in Rangoon, and the only supply at these times was an inferior quality, *left over from the last crop* [writer's italics]. It can be imagined that the journey by steamer and open boat, and subsequent storage at the mines, would not improve its quality.

'From subsequent inquiries I have ascertained that it is a fact that most of the Rangoon rice coming to the Straits is of the worst quality; it is badly cleaned and rapidly becomes fungus-grown, and in the latter months of the year has deteriorated to such an extent that (as a large importer of this rice told me the other day) "it is fit only for the feeding of the Straits mining coolies."

'We have, then, at Sungei Lembing a large mining centre which has been opened up for many years, but at which beri-beri shows no signs of becoming less prevalent or virulent in type, conditions which are very unusual, if not, so far as the rest of Malaya is concerned, unknown.

'How is this apparent anomaly to be explained? I would suggest that it may be due to the fact that this particular centre of mining has not yet been brought into quicker communication with the source of supplies. It is, for transport facilities, in the same backward position that it was in ten years ago. The coolies are still fed with stale and mouldy uncured rice, as surely as the north-east monsoon comes round.'¹

¹ Dr. Lucy goes on to add the following :

'Wherever we have a new district or mine opened up in a remote place, there also we have delays in transport; and these delays mean deterioration of the rice-supply. At first delays are very long; gradually, as roads are made or rivers cleared, they become shorter; the rice is got up to the mines in better condition, and beri-beri decreases with improved transport.

'A good example of these conditions occurred at Bentong, in Pahang.

In this case the increased toxicity of the rice may have been—most probably was—wholly acquired before it was delivered at the centre where it produced its effects. And unless the rice used has also been grown locally it must always be the case that the original infection of it, and such virulence of its *noxa* as depends on this original taint, are independent of local conditions. But the latter, it must be assumed, will in all cases have also their own effect—will increase, will check, or arrest the further making of the poison.

The Sungei Lembing instance is given as a type of many where an appearance of seasonal or periodic prevalence of beri-beri is fallaciously set up by factors which are, however, purely economic.

The increasing virulence of the extrinsic factor—the rice, which more and more deteriorates—combined with variations in the intrinsic one—the unequal extent to which individuals are affected by its action—may bring about, further, another and remarkable state of affairs, for which I do not know that any parallel is afforded by the natural history of other supposedly infectious diseases, and which therefore deserves consideration.

Toleration as a Factor in Periodicity.—To make the point about to be shown more clear, a preliminary reference may be permitted to the conditions under which immunity against, or toleration for, other intoxications is acquired.

All diseases may be regarded as due to poisons, whether these are introduced ready-made into the system from without, or whether they are elaborated by germs or abnormal processes within.

Against most diseases it is, again, certain, and against all it is

Before the trunk-road was made, the tin-mining coolies at this place were decimated by the most virulent form of beri-beri. In one epidemic over 70 per cent. of the cases were fatal, and hundreds died of the disease. At this time the coolies' rice was brought chiefly *via* the east coast, up river; it was several weeks in transit, during which time it was more or less exposed to the weather in open boats. The rice was brought up in very large quantity, and stored at Bentong. Since the completion of the trunk-road rice has been conveyed to Bentong entirely by bullock-cart from Kwala Kubu, which is on the railway. No large supplies are now stored at Bentong, and beri-beri is no longer markedly prevalent or severe in type in that district. Many such instances might be quoted where a newly-opened-up mine in a remote district is scourged with beri-beri. Transport facilities increasing, the necessity for the storage of rice in large quantities and for prolonged periods disappears, and beri-beri assumes its normal proportion.'

highly probable, that immunity may be gained by gradual training of the organism to resist. Essentially the process seems to be in all cases that for every unit of poison presented there is produced that which will destroy or neutralize it—an 'antibody' or antidote. But the unit of poison first given must be no more than sufficient antibody already exists for, or can be called forth to neutralize, within the system. The process once begun, by gradual stages a condition is reached in which there is sufficient antibody to meet all the poison which is likely to be presented, or (possibly) which can be absorbed. In this way the immunized horse resists doses of cobra or diphtheria venom; the arsenic, the opium, the alcohol habitués resist doses of these poisons many times greater than would have been fatal had they been the first doses absorbed.

But in such a process the graduation of the dose, varying for different individuals, is of importance. It cannot at any stage be suddenly increased. If its application, again, be intermitted, the resistance before accumulated is impaired and may be lost.

What is the bearing of these reflections upon beri-beri? It is obvious that if beri-beri be due to the absorption of a poison from rice, the production of its epidemics must depend largely—

1. On the extent to which those made to consume a given supposed toxic sample of the grain are *used* to rice of that description.

2. On the extent to which the rice which any given community is made to consume is toxic when *first* they are made to consume it.

As with other poisons, so with that causing beri-beri it must be supposed that constant consumption will induce growing toleration for its effects; so that doses may ultimately be withstood—be absorbed apparently without producing any ill-effect whatever—which, had they been first or more rapidly taken, must have caused symptoms.

Supposing a community to be supplied with rice which, at first not at all, or only slightly noxious, gradually becomes more so, until at last it is highly toxic, the individuals habituated to its use may by that time have acquired such toleration that few or none of them show signs of its action (beri-beri).

But suppose that at the juncture when the rice used has reached its greatest toxicity (or such a degree of it as to entail beri-beri in some) fresh individuals join the community who are unused to rice in this condition, who consequently have no toleration for, or immunity against, its effects. Will not the result naturally

be that these new arrivals will, if not all and immediately, yet many of them and rapidly, succumb to the poison ?

Just such a happening as this is illustrated by an observation of H. Wright's, the facts as to which I now give direct from his work, although he himself uses them for quite another conclusion.

'79. . . . In an inquiry,' says this writer ('Study,' etc., p. 10), 'at various mining centres where sinkhehs are mostly employed I find that if they are brought into the country during the north-east or wet monsoon, and sent to a mine where beri-beri is rife, many of the new-comers acquire the disease within a month, a smaller number within a fortnight, and a few within ten days.

'80. If brought in during the south-west or drier monsoon the disease is seldom contracted so early, not until the wetter north-east monsoon begins to blow.'

Wright analyzes 276 inquiries made under this head, with results (rearranged by the writer) as follows ('Study,' Table I., p. 11) :

Of 142 Sinkhehs arriving within First Month of South-west Monsoon—i.e., in April, there got Beri-beri					Of 132 Sinkhehs arriving during First Month of North-east Monsoon—i.e., in September, there got Beri-beri				
Within 10 days,		nil—i.e.,		in May	Within 10 days,		12	(Sept.)	
" 14 "		nil	"	May	" 14 "		32	(Sept.)	
" 30 "		nil	"	May	" 30 "		34	(Sept.)	
" 2 months,	1		"	June	" 2 months	} 42	(Oct.)		
" 3 "	3		"	July	" 3 "		(Nov.)		
" 4 "	2		"	Aug.	" 6 "		14 (Dec. to Feb.)		
" 5 "	20		"	Sept.					
" 6 "	32		"	Oct.					
" 7 "	43		"	Nov.					
" 8 "	41		"	Dec.					

Assuming the correctness of Wright's information, the fact appears clearly established by his figures that coolies arriving in autumn incur beri-beri more rapidly than they who arrive in spring.

Wright considers the table proves that the incubation period of beri-beri is (at any rate in the case of people who reach 'infected centres' in autumn) a short one; why it should be so much longer in the case of spring arrivals is not made clear. But of the really remarkable fact that the later comers should succumb almost immediately to beri-beri, at places where others of their own class, but who had arrived earlier, had resisted for so many

months, and, what is more, even continued for the most part to resist, while the last arrivals were severely suffering—for the table shows this also—of this really extraordinary phenomenon no explanation is offered by Wright.

It is true he suggests the vague influence of weather: 'The direction of the monsoons appears to be a determining factor in beri-beri' (*loc. cit.*, par. 51)—an ascription which seems to have been prompted by that ineradicable old-world tendency which has attributed so many mysterious maladies to the moon. But the circumstances rather offer an excellent illustration of the position which has been already predicated.

Lucy's evidence has shown that the rice consumed generally throughout the country by Sinkhehs, bad to begin with, becomes progressively more and more deteriorated as the year moves on.

Coolies, therefore, who first arrive in the country toward the end of the year are thrown at once upon consumption of it in its most toxic condition, hence quickly evincing its effects.

Those who arrive earlier, in spring, on the other hand, are at that time given rice comparatively new, the poison in which, then slight, they are able to resist, their toleration for it increasing *pari passu* with its increasing virulence. In the end, when the *noxa* in the rice has reached an intensity which speedily prostrates new-comers, these earlier arrivals, as has been seen, are able to withstand its effects far longer than the new arrivals, and often are completely proof against its action.

The influence of factors such as have just been dwelt upon in determining what are apparently purely seasonal movements of beri-beri does not, however, exclude that of local physical changes too. Our position is merely that the former may on occasion increase or even over-ride the local factors.

Although in the writer's view the conditions which *chiefly* determine the production of epidemics, especially the meteorological ones, are not the local ones prevailing when the actual outbreaks take place, but others often long antecedent, there can be no doubt that both weather and other factors (such as mode of storing rice), operating where the material is used, are potent for evil too.

As in the Malay Peninsula, so all over the world discrepant accounts as to the seasons at which beri-beri most occurs have been given by different observers.

The observations connecting greater beri-beri incidence with

definitely *wetter* seasons are most numerous, and some of them are such as it seems impossible to set aside.

Thus Reaucar¹ states that at Poulo-Condore cases were always most numerous in the hotter, which were also the most humid months—those of July and August, the end of the south-west monsoon. Pekelharing and Winkler's careful observations² showed that at Atjeh there were two waves of increased prevalence, one at each of the two rainier epochs—April to May and September to October—during each of the three years, 1886 to 1888, and this was the case both with the European troops, the Amboinese, the native (Malay) troops, and the convicts.³

In the Java gaols, Verderman⁴ says, it is notorious that beri-beri is always more prevalent during the later, and there rainier, months of the year. Leslie⁵ observes the same of the outbreaks in Burmah gaols. He relates that at the S.P.C.K. Mission School at Bassein cases occurred regularly every rainy season for six years, while during the months of July, August, and September there were no cases.

In none of these examples, however, had the influence whether of the age of the rice or of other economic factors been considered, so that it is possible that in them, too, a periodicity apparently entirely seasonal may have really depended upon other things.

Multi-annual Changes.—The fallacies attending comparison of beri-beri with seasonal changes within the year, which must obscure any real relationship to climate (if such exist) disappear when the conditions obtaining at different places during a succession of years are compared.

The writer has made a careful study of records of admission for beri-beri and other diseases, and of the rainfall at thirty-five different district hospitals and infirmaries between 1880 and 1902, and believes that the following conclusions may be accepted :

1. *There is no definite relation between rainfall and beri-beri in Malaya.*

In the table and chart attached the rainfall and beri-beri prevalence are exhibited in relation to *thirty-one different districts*.

¹ 'Thèse,' p. 23. ² 'Beri-beri' (Bentley's translation), p. 147, and charts.

³ Although extensive disinfection was practised at Atjeh, with the effect (as these authors believed) of diminishing the general incidence of the disorder, it is notable that both during and in the period following the disinfection these peculiar exacerbations recurred at the same junctures and to the same relative extent as before.

⁴ 'Onderzoek,' p. 120.

⁵ 'Notes and Statistics Hosp. and Disp., Burmah,' 1899.

336 THE CAUSE AND PREVENTION OF BERI-BERI

The table represents an analysis of 328,936 admissions to thirty-one district hospitals during the seven years 1895-1902. Of these the Chinese formed 73 per cent. of all cases, and contributed the whole¹ of the beri-beri.

The cases of beri-beri numbered 57,025. The places are arranged in order of relatively greater rainfall, as observed at each hospital.

EXPLANATION OF CHART.

1. The thirty-one stations at which observations were taken are arranged from left to right in order of greater mean annual rainfall as observed during seven years—1896-1902.

2. The space coloured yellow represents the rainfall. Each $\frac{1}{2}$ millimetre of ordinate represents 1 inch rainfall.

3. The curves represent respectively the mean of seven years' observations, at each station, of—

(a) Case-incidence of beri-beri, or proportion of cases of beri-beri, per 1,000 Chinese patients admitted to hospital.

(b) Case-mortality of all beri-berics admitted, or deaths per 1,000 admissions.

(c) Relative general sickness of Chinese compared with other races, measured by the proportion of Chinese admitted for all other diseases, exclusive of beri-beri, per 1,000 of all patients admitted.

The scale of all three curves is the same, and is such that $\frac{1}{2}$ millimetre of ordinate represents a movement (up or down) of 5 per 1,000, or $\frac{1}{2}$ per cent., in the ratios.

The abscissa line through each curve, and the number stated on it, represents the average case-incidence per 1,000 at the place for the whole period.

For convenience the three scales are superposed, so that the mean of all stations in regard to each of the three factors coincides.

It will be seen from the table, and more clearly from the chart, that with a rainfall varying between 68.85 inches and 166.28 inches annually, and a case-incidence of beri-beri of between 17 and 472 per 1,000, there is *no indication of any movement in common*. Both the prevalence of beri-beri is high where the rainfall is low, and the converse, and low or high rates of either may go together. As it could be contended that the influence of rainfall is obscured,

¹ Since, as shown in table, 97.6 per cent. of the cases of beri-beri admitted to hospitals are Chinese, even in the Straits Settlements, where the proportion of other nationalities is greatest, the discrepancy, amounting to less than 3 per cent., has been ignored, and all the cases of beri-beri taken as Chinese.

PERIODIC VARIATIONS

Telebu	
Kuala Selar	
Kuala Kang	
Tampin	
Jasin	
Seremban	
Kuala Lang	
Malacca	
Kajang	
Sungei Besi	
Klang	
Bagan Sera	
Alor Gajah	
Parit Buntar	
Teluk Anson	
Batu Gajah	
Kuala Lum	
Butterworth	
Ipoh	
Sungei Bak	
Bukit Mert	
Gopeng	
Serendah	
Kuala Kub	
Penang	
Balik Pulau	
Rawang	
Tapah (five	
Selama	
Kampar	
Larut	
All sta	

¹ Since, as shown in table, 97·6 per cent. of the cases of beri-beri admitted to hospitals are Chinese, even in the Straits Settlements, where the proportion of other nationalities is greatest, the discrepancy, amounting to less than 3 per cent., has been ignored, and all the cases of beri-beri taken as Chinese.

DISTRIBUTION OF BERI-BERI IN BRITISH MALAYA.
Rainfall and Prevalence of Beri-beri at Thirty-one District Hospitals for Seven Years (1895-1902).

Place.	Actual Total.				Average Rain-fall in Inches Annually.	Proportion per 1,000 of		
	Admissions for all Causes (all Races).	Admissions of Chinese.	Admissions for Beri-beri.	Deaths from Beri-beri.		Cases of Beri-beri to all Chinese Admissions.	Admission of Chinese to all, excluding Beri-beri.	Deaths to Cases of Beri-beri.
Jejebu ..	2,924	2,385	962	41	68.85	403	726	42
Kuala Selangor ..	1,430	438	23	7	74.66	52	294	303
Kuala Kangsar ..	11,545	5,542	624	45	81.07	113	450	72
Tampin ..	3,397	2,698	316	58	82.72	117	773	183
Jasin ..	6,851	6,353	903	157	85.99	142	906	173
Seremban ..	12,860	7,169	3,385	230	88.92	472	399	68
Kuala Langat ..	1,903	769	131	51	89.66	170	360	389
Malacca ..	16,687	14,494	2,182	394	92.12	150	849	180
Kajang ..	8,227	4,953	1,786	144	92.78	360	491	80
Sungei Besi ..	1,714	1,626	747	122	92.78	459	908	163
Klang ..	7,554	3,397	379	56	93.39	111	420	147
Bagan Serai ..	14,358	10,521	214	20	95.27	21	728	93
Alor Gajah ..	6,531	4,694	824	54	95.72	173	677	148
Parit Buntar ..	4,285	2,361	58	19	96.16	24	544	328
Teluk Anson ..	5,652	1,999	315	72	98.51	152	315	228
Batu Gajah ..	26,670	21,322	8,938	1,220	98.54	419	692	136
Kuala Lumpur ..	33,735	27,687	8,422	1,702	99.92	304	761	202
Butterworth ..	4,824	1,564	139	33	101.14	89	304	238
Ipoh ..	24,653	22,012	7,643	1,128	101.98	347	844	147
Sungei Bakap ..	3,766	1,062	18	4	101.99	17	278	222
Bukit Mertajam ..	3,504	2,599	124	29	105.65	47	760	233
Gopeng ..	16,781	14,189	4,319	522	111.71	304	792	128
Serendah ..	10,185	9,375	2,807	442	114.57	299	890	157
Kuala Kubu ..	15,892	9,935	2,019	487	115.38	203	570	241
Penang ..	25,533	16,622	2,093	721	116.52	126	570	344
Balik Pulau ..	3,588	2,785	183	57	120.46	65	764	311
Rawang ..	7,075	6,410	1,897	302	135.44	296	871	159
Tapah (five years only) ..	12,147	5,621	1,704	338	136.22	303	375	198
Selama ..	404	173	26	6	140.12	150	389	231
Kampar ..	8,082	7,386	2,115	173	146.59	286	883	82
Larut ..	27,326	18,897	1,729	356	166.28	91	670	205
All stations ..	328,936	231,418	57,025	8,990	110.74	246	734	158

¹ Since, as shown in table, 97·6 per cent. of the cases of beri-beri admitted to hospitals are Chinese, even in the Straits Settlements, where the proportion of other nationalities is greatest, the discrepancy, amounting to less than 3 per cent., has been ignored, and all the cases of beri-beri taken as Chinese.

Place.	Actual Total.				Average Rain-fall in Inches Annually.	Proportion per 1,000 of		
	Admissions for all Causes (all Races).	Admissions of Chinese.	Admissions for Beri-beri.	Deaths from Beri-beri.		Cases of Beri-beri to all Chinese Admissions.	Admission of Chinese to all, excluding Beri-beri.	Deaths to Cases of Beri-beri.
Jebebu ..	2,924	2,385	962	41	68.85	403	726	42
Kuala Selangor ..	1,430	438	23	7	74.66	52	294	303
Kuala Kangsar ..	11,545	5,542	624	45	81.07	113	450	72
Tampin ..	3,397	2,698	316	58	82.72	117	773	183
Jasin ..	6,851	6,353	903	157	85.99	142	906	173
Seremban ..	12,860	7,169	3,385	230	88.92	472	399	68
Kuala Langat ..	1,903	769	131	51	89.66	170	360	389
Malacca ..	16,687	14,494	2,182	394	92.12	150	849	180
Kajang ..	8,227	4,953	1,786	144	92.78	360	491	80
Sungei Besi ..	1,714	1,626	747	122	92.78	459	908	163
Klang ..	7,554	3,397	379	56	93.39	111	420	147
Bagan Serai ..	14,358	10,521	214	20	95.27	21	728	93
Alor Gajah ..	6,531	4,694	824	54	95.72	173	677	148
Parit Buntar ..	4,285	2,361	58	19	96.16	24	544	328
Teluk Anson ..	5,652	1,999	315	72	98.51	152	315	228
Batu Gajah ..	26,670	21,322	8,938	1,220	98.54	419	692	136
Kuala Lumpur ..	33,735	27,687	8,422	1,702	99.92	304	761	202
Butterworth ..	4,824	1,564	139	33	101.14	89	304	238
Ipoh ..	24,653	22,012	7,643	1,128	101.98	347	844	147
Sungei Bakap ..	3,766	1,062	18	4	101.99	17	278	222
Bukit Mertajam ..	3,504	2,599	124	29	105.65	47	760	233
Gopeng ..	16,781	14,189	4,319	522	111.71	304	792	128
Serendah ..	10,185	9,375	2,807	442	114.57	299	890	157
Kuala Kubu ..	15,892	9,935	2,019	487	115.38	203	570	241
Penang ..	25,533	16,622	2,093	721	116.52	126	570	344
Balik Pulau ..	3,588	2,785	183	57	120.46	65	764	311
Rawang ..	7,075	6,410	1,897	302	135.44	296	871	159
Tapah (five years only) ..	12,147	5,621	1,704	338	136.22	303	375	198
Selama ..	404	173	26	6	140.12	150	389	231
Kampar ..	8,082	7,386	2,115	173	146.59	286	883	82
Larut ..	27,326	18,897	1,729	356	166.28	91	670	205
All stations ..	328,936	231,418	57,025	8,990	110.74	246	734	158

in different localities, by variations in the proportion of 'material for infection'—*i.e.*, in the number of Chinese admitted to the hospitals, I have added an analysis of the relative proportion of this race to other nationalities admitted at the same time at each hospital for *all diseases excluding beri-beri*. It would seem that neither does this factor by itself affect the greater or less prevalence of beri-beri. The whole population nearly of a hospital (at Jasin 90 per cent.) may be Chinese, and yet the incidence of beri-beri be very low (14 per cent. in the case cited). In the place where there was least beri-beri (Sungei Bakap, in the province Wellesley—17 per 1,000 only), 25 per cent. of the patients were Chinese. At Larut, where the case-incidence was 65 per 1,000, over two-thirds of the patients were Chinese—and so on.

As pointed out earlier in the work, among Chinese it is chiefly the sinkhehs who furnish the cases. Still, the proportions of laukhehs and free coolies who get the disease also is not small, and since in such places as Singapore, Penang, Malacca, where laukhehs and free coolies form the greater part of the Chinese population, beri-beri is severely prevalent, it follows that even entire absence of sinkhehs will not prevent the disease becoming evident when the cause of it is abroad in sufficiently intense form. Yet it is probable that, when the toxic effect of rice is but slight, results among laukhehs may fail to appear, so that in some places slight waves of increase will fail to be represented. Also, laukhehs taking, by reason of their better feeding, longer to evince effects, it is likely that, in places where they are the chief material for beri-beri, the influence even of a more than usually toxic rice, though ultimately apparent, will be delayed.

In this way a wave of increase affecting one place in one year may at another place not be represented till the year after. Such sources of error may account for absence of, or delay in, any increase at particular places, but will not prevent its occurrence at places where the most susceptible material (sinkhehs) is known to exist. For this reason I have marked on the charts the character of the districts concerned, and referred in each to the principal occupation of the inhabitants. In all those marked as 'Mining' it may be taken for granted there is a large proportion of sinkhehs.

In regard to the case-mortality of the disease, a similar absence of conformity with other factors is to be observed. It bears relation neither to the rainfall nor even the prevalence of the disorder. At places where either of these factors may be greater the mortality may be less, and conversely.

In order to place these conclusions on as extensive a basis as possible, I have analyzed all the admissions of which I have been able to gain records at the different hospitals—thirty-six in number—of which the statistics are given in tables in the Appendix. Of the eighteen principal ones among these, graphic records are attached. This more extended examination confirms the conclusion stated. While, as follows from the probabilities, some of the fluctuations in both rainfall and case-incidence agree, they even oftener tend in opposite directions.

Observations of rainfall were made at twenty-six of the stations for which records of admissions of beri-beri are also available. The total occasions of comparison—year's total rainfall and year's case-incidence of beri-beri—are 349. Upon 160 of these—45 per cent.—the movements—rise or fall of either factor as compared with the preceding year—agreed. Upon the remaining 55 per cent. they were divergent. The case-mortality and the case-incidence were compared on 439 occasions. On 47 per cent. of these there was agreement in direction, in 53 per cent. disagreement.

No causal relation, it may safely be asserted, then, exists between the fluctuations of beri-beri and the rainfall *in the Malay Peninsula*.

2. *Though independent of meteorological factors, the local variations in beri-beri incidence nevertheless themselves coincide in time at different stations.*

Taken year by year, the changes—increase or decrease of prevalence—at each station agreed in direction, though not in extent, on at least two out of every three occasions, upon which the incidence for the year was compared, at the thirty-one different places, during twenty-one years analyzed in the table preceding. If two-year periods be taken for comparison, so that waves which occur—as often happens—at the end of one and the beginning of another year may be treated singly, the correspondence of the variations is seen to be extraordinarily close.

Charts are attached showing the period and extent of the fluctuations at the twenty-seven district hospitals at which the admissions were most numerous.

The fact which these charts clearly illustrate is that each of the various waves of increase or decrease, which took place in the prevalence of beri-beri between 1880 and 1902, affected almost every district in which observations were made, and almost simultaneously.

340 THE CAUSE AND PREVENTION OF BERI-BERI

During this period the analysis indicates six such principal waves of increase happened, which may be tabulated as follows :

FLUCTUATIONS IN BERI-BERI INCIDENCE.

No. of Observations.	Year.	Nature of Movement.	No. of Stations.	No. in Agreement.	Remarks.
I. (a)	1881-1882	Great increase	2	2	Larut and Malacca. This was part of the great wave observed at Mamila.
(b)	1882-1883 ¹ / ₂	Decline	4	3	The exceptional station in 1883 was Seremban, at which an increase noted probably apparent only, as the hospital was opened that year.
(c)	1883-1884	Decline continued	9	6	Of the exceptions, one, Kuala Lumpur District Hospital was opened in that year; at two other stations the following rise (d) was anticipated.
II. (d)	1884-1885	Rise	11	8	Exceptions, Kuala Lumpur Prison Infirmary, Klang, and Larut, where, however, the incidence still remained very high. At Larut the material, sinkhehs may have been diminishing.
(e)	1886	Rise continued or fall began	10	5 (rose)	—
(f)	1887		13	5 (fell)	
		Fall		10	Exceptions: Malacca and Kuala Lumpur Gaol, where fall began 1886; and Seremban, where following rise was anticipated (e).
III. (g)	1888-1889	Rise	13	10	Exceptions: Seremban (falling after 1887), Penang Pauper Hospital (rise of 1890 anticipated), and Kuala Kangsar, at which incidence remained high.
(h)	1890-1891	Fall	20	15	Exceptions: Klang, Kajang, Penang Pauper, Kuala Lumpur District, Jelebu, all of which anticipated the rise following.
IV. (i)	1892-1893	Small rise	22	17	At the five exceptions the rise was anticipated in (h).
(k)	1894	Fall	22	15	In seven exceptions the fall had preceded in two and followed in five (in 1895).
V. (l)	1895-1896	Rise	27	24	One (Kajang) continued the fall of 1894, but rose in 1897.
(m)	1897	Fall	27	24	Three exceptions anticipated rise of 1899.
VI. (n)	1899-1900	Rise	27	20	One remained level; in six decline of 1900-1901 (o) began.
(o)	1901	Decline	27	21	—
(p)	1901-1902	Decline	27	20	—

of
at,
ed
ere
re-
er,

he
ts,
use
he
ns

se
k-
ed
he
of
to
ny
on

of
w,
is
ns
ns
ter

ice

he
ch

for
the
to

increase or decrease *simultaneously* year after year at each of thirty stations, separated for the most part by scores of miles of unbroken jungle and holding no communication of common traffic?

But the knowledge that rice is the source of the disease, and that this article is obtained from common areas and distributed usually at the same time to each place, makes the understanding of the phenomenon easy.

the rice is grown. There, it is likely enough, variability in its intensity will be found to depend on rainfall, and other agencies which favour or hinder the development of a living agent affecting the corn.

That the periodic movements of beri-beri in the peninsula are due to an extra-regional influence is a view which is confirmed by more extended observations.

Not only at the various stations of British Malaya, but in almost all the countries where beri-beri is endemic, and in places beyond them where epidemics have occurred, the exacerbations of the disease may be observed to be nearly simultaneous.

Thus, of the six marked waves of increase of beri-beri occurring in the Native Malay States, as noted above :

I. *a, b* (1881-1882).—Corresponded nearly with epidemics in the Singapore gaols (1878-1884), at Poulo-Condore (1879-1881), a marked increase in the Japanese Navy (1882), and the great outbreak in Manila (1881-1882).

II. *d* (1884-1885).—Corresponded with increased incidence in Dutch Army and Navy at Atjeh, in German East Indies, at the Cocoskeeling Islands, in Burmah (Mandalay and other gaols), in the Soudan, and on shipboard in England (Chinese cruiser *Toonan* at Newcastle) and New York (? Newfoundland fishermen).

III. *g, h* (1888-1889).—Corresponded with a great epidemic on the Belgian Congo.

IV. *i, k* (1892-1893).—In New Caledonia (1891, 1892), in the Dutch Navy, cases in Newfoundland (1890), and in France (Nantes), and numerous ships in English ports or at sea, mentioned by Rees, Orr, Jameson, and others. The famous epidemic at the Richmond Asylum, Dublin, began in autumn, 1894.

V. *l* (1895-1896). 1895.—Epidemics in *prisons*: Pudo, Selangor (began 1895, continued more or less to 1903); Dakar, Senegal (negroes); Wyndham, Australia (aborigines). At *asylums*: Melton, England; Little Rock, Arkansas; Tuscaloosa, Alabama; Rangoon, after seven years' freedom. Cases on land in Australia (Chinese, Melbourne, Sydney), in Kurile Isles (Shimushu, Japanese), numerous ships (mentioned by Rees, Roll, and others).

m, 1896.—Richmond Asylum (after a year's freedom), and at

Tuscaloosa, and Rangoon, ships of Rees's fleet, ships at Cork, Falmouth, and Glasgow and Java prisons.

m (1897).—Richmond Asylum, and one at St. Gemmes-sur-Loire, Madras Regiments in India, German New Guinea, Cuba, and St. Helena; Singapore Prison (after twelve years' freedom, continued to date), numerous ships (*City of London* at Glasgow, *Chusan* at Colombo, a schooner at Dublin), others at Falmouth and in London.

1898.—Dublin, Ascension, Madras, Poulo-Condore, Bangkok, and on ships (London, Falmouth, Red Sea, Para, etc.).

VI. *n, o* (1899-1900).—Burmah (several gaols), Dublin, Annam (Chantabun), Ni-i-gata Prison (Japan) in 1899; at Hong Kong (foundling homes), Dublin, sporadic cases in British Guiana, Egypt; many ships (H.M.S. *Sphynx*, Indian Ocean; Falmouth—three ships).

p (1901-1902).—St. Helena (epidemy among Boer prisoners), twenty-one vessels touching at British ports, South Africa.

At several of the periods of increased beri-beri reported cases of *peripheral neuritis* also seemed to have been more numerous. In 1884 there was reported by Broadbent¹ a series of five 'alcoholic' cases, and by Hadden² and others; in 1893 three cases termed 'puerperal' by Möbius³ and Luntz⁴; in 1895 a series of ten or eleven cases following chancroid, but not syphilitic, by Maitland⁵ in India; in 1897 two cases at the Grafenburg Asylum, West Germany (Oerthmann)⁶; and some in Saxony (Alt Scherlitz Asylum in Leipzig). These latter Norman⁷ has referred to as beri-beri, but Scheube⁸ demurs to this description.

Ucherman gives figures showing a rapid and surprising increase in the deaths ascribed to beri-beri among Norwegian sailors from 1893 onwards. He quotes Stian Erichsen as saying that 'Before 1890 the disease was unknown among our seamen.' From 1893 to 1898 cases occurred on no less than twenty small sailing-ships trading between Tvedestrand and Arendal and various tropical ports.

¹ *Proceedings of the Royal Medico-Chirurgical Society*, February 12, 1884.

² *Proceedings of the Pathological Society*, October 25, 1884.

³ *Münich Med. Woch.*, No. 8, 1892.

⁴ *Deut. Med. Woch.*, November 22, 1894.

⁵ *British Medical Journal*, February 4, 1890, i. 290.

⁶ *Algm. l. f. Psych.*, May 18, 1898, Bd. lv., 1, p. 84.

⁷ *British Medical Journal*, September 24, 1898, ii., p. 873.

⁸ *Arch. f. Schiffs u. Trop. Hyg.*, 1898, No. 6, p. 329.

344 THE CAUSE AND PREVENTION OF BERI-BERI

Of 2,393 deaths of Norwegian sailors abroad, in six years, beri-beri was accountable for the following :

1893	1	1896	20
1894	0	1897	25
1895	4	1898	16

Prior to 1884 the syndrome of symptoms now called *multiple peripheral neuritis* was so rare (we are forced to conclude) that it had not been distinguished, or at all events had not received authoritative recognition. Magnan (*L'Alcoolisme*, 1874), though describing cases of paralysis due to alcohol, had not separated them from paraplegia. Boehm (*vide* Ziemmsen's 'Cyclopædia,' 1878) failed to make note of any alcoholic paralysis. So brilliant a clinician even as Fagge (*Prin. and Pract. Med.*, 1886), and his accomplished editor, Pye-Smith, gave multiple peripheral neuritis no place in their nosology. But in 1884 cases of general peripheral neuritis were described (at the Royal Medico-Chirurgical Society, February 12) by Broadbent, and referred to alcohol. In the discussion on these cases Wilks recalled similar ones which had recovered, and Buzzard stated that other such had been brought before the Society by Dr. Reginald Thompson in 1868.

In *Brain*, vol. vii., 1884, Dreschfield gave an account of more alcoholic cases, seen in Manchester. He observed that the complex had first been described by Dr. J. Jackson, U.S.A., in 1822.

The classical account of Ross appeared in 1890 (*Medical Chronicle*, March and October, 1891), and was derived from observation of some 13 cases seen in or about Manchester, and 77 collected from medical literature. Thereafter cases became apparently everywhere increasingly numerous—at all events, they were more often described, and many, following in the wake of various illnesses, were imputed to these illnesses as their cause. A list of at least thirty such supposed effective causes of peripheral neuritis is now recognised, and chief among them is arsenic.

Thorburn and Williamson state that at Edinburgh from 1892 to 1897 nearly 6 per 1,000 of all in-patients treated in the infirmary were cases of peripheral neuritis (alcoholic); in London, during five years—1894 to 1898—4.5 per 1,000 of all in-patients at one hospital (St. Thomas's) were peripheral neuritis, probably mostly alcoholic.

In and around Manchester and other cities towards the

North of England cases of neuritis, presumed to be alcoholic, had long been more numerous than elsewhere. Tattersall shows that the *deaths* recorded as due to this complaint, which prior to 1893 were but 2 or 3 annually, began to rise in 1893; rapidly increased 1899 to 1900, in the last and first half-years of which there were 20 such deaths; and reached a climax in the second half-year of 1900, when 50 deaths took place from this single complaint. These figures refer to a single borough—Salford.

In the year of greatest incidence most of the cases happened in the autumn—August to November. Towards the end of 1900 Dr. E. S. Reynolds,¹ having detected the presence of arsenic in the beer which most of the sufferers from the disease had been in the habit of drinking, the source of the malady was attributed to that cause.

It is not only the occurrence of so many scattered epidemics at or about the same intervals which is remarkable. The *absence* of similar records at intervening periods is not less so.

It may be said that some of these epidemics were not beri-beri, but peripheral neuritis of some other origin. But since, in the dubious cases, it is not for clinical, but epidemiological, reasons that their diagnosis is questioned, their concurrence with so many other outbreaks of a disease clinically almost identical at least affords the presumption that they, too, owned similar origin. Allowing for the various sources of delay which distance and economic factors oppose to the access of any morbid agency to different communities, the near coincidence of so many epidemics of beri-beri in point of time, again, justifies the conclusion that all had not only a similar but the same origin—that they were derived actually from one and the same common source.

This result, it may be pointed out, alone would oppose the conception of the disease as being due to infection. For what form of contagion could be supposed to *simultaneously attack*—and not once, but repeatedly—communities so widely separated as the asylums at Rangoon and Dublin, France and Alabama? Or prisons in Singapore and Senegal, places between which there is absolutely no communication?

It may be suggested that *influenza* has at times spread rapidly enough over the world to make its effects appear apparently simultaneous in different hemispheres. But the march of this malady is marked by victims of the contagion, who, if they are not actually its only transmitters, at least provide evidence of the intermediate stages of its progress from one point to another.

In the case of beri-beri the infection-hypothesis forces its adherents into the

¹ *British Medical Journal*, November 24 and December 22, 1900.

position that one common contagion affects simultaneously quite limited foci, separated by enormous distances, without any direct movement of persons between them and through the agency of individuals themselves always unaffected.

Not to slay, but to demonstrate once again the death of the already slain, the fact may be recalled, offering fatal opposition to any theory of causation of beri-beri, whether by contagion or air-borne infection, that the incidence of the disease on mixed communities is *selective*, following differences of food, which obviously no air- or person-infection could possibly be.

Equally does the variation in the intensity of the original cause of beri-beri, independently of conditions of the place where its effects are observed, militate against the nitrogen- or fat-starvation, or other purely dietetic theory of its production. For it cannot be supposed either that the proportion of one or another of the components of diets should be changed always in the same way or in the same degree at the same time by the many different peoples whom beri-beri affects, and always at the exact junctures when the influence of the disease becomes more intense also upon persons whose diets have undergone no such change.

Of what nature could a morbid agent be, the influence of which should be nearly simultaneously felt in those places which it has been shown so to affect?

In these days of rapid ocean transit a cargo of rice produced in Siam in July or August, and milled in Bangkok in September or October, may be distributed in November or December between Japan and Australia, England and South America. There is no difficulty, therefore, in explaining the possible distribution of a disease derived from rice almost simultaneously all over the world wherever there are rice-eaters to be affected.

Again, as with ergot, the smuts and bunts and other epiphytes and parasites of grain, it is the case that in whatever region the grain is grown there also its parasites are likely to be found. It follows that wherever rice is grown the postulated beri-beri agent which affects it is almost sure to be found also. Not only so, but since of the class of pests referred to, although all affect by preference particular cereals, there is none which may not at times affect others, the beri-beri agent will, as has already been predicated by the theory, be likely to affect at times other grain.

In the possibility of such an occurrence may be found the explanation of many cases of beri-beri in which the operation of rice is not traceable.

The connection which these facts have with the periodicity of beri-beri is this: that as there is an *optimum* concurrence of conditions of growth for the plant or its epiphyte, so there must be a *pessimum*. The fluctuation of these two will determine the

periodicity of the disease. But since, wherever the host or the parasite is cultivated, the conditions must be nearly the same, and since large periodic movements of climatic change are world-wide, it is likely that the *optimum* conditions for the growth of the beri-beri agent will also happen everywhere nearly simultaneously. The effect—the disease—naturally following the production of the agent, will therefore appear at most places during the same years.

The demonstration of irregular periodic changes in the production of the beri-beri agent is important as explaining both the sudden appearance and disappearance of the disease under circumstances in which no other change is observable. Thus it is obvious how, for this reason, it must occur that persons who have for a long time been in the habit of eating a given quantity of one sort of rice without harm should at another, when the actual sample is derived from a fresh crop containing more, or more virulent poison, fall victims to beri-beri.

Since the rice harvest in the main countries of its supply is at the end of summer, the new crop then reaped, and milled in autumn, comes into consumption usually about the beginning of the next year. It is about this juncture, therefore, that it might be expected to be found beri-beri epidemics should most commonly be liable to *termination*. There seems to have been such a happening in the instance which Durham¹ notes, where the disease, which had for many years severely affected four places—the prisons of Singapore and Kuala Lumpur, the lunatic asylum at Singapore, and Christmas Island—disappeared abruptly from each in the same month—January, 1903, only to recur, however, at each place later on.

Many recorded instances of the supposed good effect of various measures in checking beri-beri doubtless owe their explanation to the same cause. The unusual intensity in the agent declines, and the disease ceases. Then, *post* and *propter*. In German New Guinea, when beri-beri was very prevalent, it was projected to build a sanatorium for the European sick, but just when this was about to be erected suddenly the disease disappeared.

At the Cocos Islands beri-beri, which had been prevalent from 1881 to 1883, ceased in the latter year, and has not since reappeared.² A *breakwater* having just then been constructed with a view to saving the native quarters from inroads of the sea,

¹ *Journal of Hygiene*, January, 1904, p. 142.

² Morris, H. C. L., ref. *British Medical Journal*, 1897, vol. ii., p. 500.

which was supposed to influence beri-beri by making the dwellings damp, the disappearance of the disease was ascribed to the breakwater. In very many instances the decline or abrupt cessation of the malady, coinciding with the application of disinfectants, has been ascribed to them. Lowson¹ describes such instances, and Hartigan² others. Yet both these observers mention, in the same communications, cases where exactly similar measures (in Lowson's case taken under his own direction) proved futile.

The occurrence of pests and blights upon grain and other crops at or about the same periods over very wide areas is historical. Such blights, moreover, spread from one country to another with a speed which rivals the propagation of influenza, and makes their action appear to be everywhere simultaneous. As a concrete example may be mentioned the *Hemileia vastatrix* or coffee-leaf disease, which, derived, as it is supposed, originally from seed coffee brought from Brazil, within two or three years ruined every plantation of coffee (*Arabica*) in Ceylon.

Kohlbrugge,³ discussing the periodicity of the disease, compares its movements to those of diphtheria, and finds in its resemblance in this respect to that and other infectious disorders a proof or evidence of its infectious nature. He gives a chart representing in graphic form the statistics of Van der Burg of the prevalence of the malady in the Dutch East Indian Army operating about Atjeh during the years 1873 to 1894, which I reproduce here.

The wave of greatest prevalence (1885-1886), it will be seen, corresponds with a similar one in the Native Malay States, indicated in the charts given above. The minor waves of 1888, 1889, 1890, and 1893, are also represented in the latter.

The later incidence of the disease upon Europeans than upon natives, upon which Kohlbrugge remarks, probably receives its explanation in the fact that the former, consuming, as is probable, always less rice—in the navy, *e.g.*, the rations of Europeans contained 500 grains of it, as opposed to 1 kilo eaten by the natives—succumbed to its effect only after a longer interval. Cases produced in natives, due to the rice supply of 1885, more rapidly developed, would fall wholly perhaps within that year; those induced in the Europeans, the slower result of smaller doses,

¹ *British Medical Journal*, 1897, vol. ii., p. 843.

² *Ibid.*, December 19, 1903, vol. ii., p. 591.

³ *Therap. Monats.*, January, 1899, p. 39.

would come to account only in the following year. Much reliance cannot, however, be placed upon the statistics as evidencing the absolute variation in the disease, in the absence of information what difference in the quantities of rice eaten in the different years by all sections there may have been. In the navy, at least, as Van Leent ¹ has recorded, there were many such alterations in the rations, all of which had some effect on the incidence of beri-beri.

¹ *Gen. Tijdschr.*, Deel xx., 5, 6, 1880.

SECTION VI

BERI-BERI IN ANIMALS : A DISEASE ALSO OF HORSES, AND FOWLS FED ON PADI OR RICE

Beri-beri likely to Affect Animals, as Ergotism does.—Although not necessarily flowing from the rice theory of beri-beri, it seems likely, judging again by the analogy of ergot, that in places where the disease is endemic or the agent of it indigenous, *animals* as well as human beings should be affected.

Epidemics of ergotism have generally been heralded by the death, with similar signs of poisoning, of live stock, especially young calves. It was by producing its peculiar effects in animals fed with ergotized grain that Thuillier, in 1630, first conclusively proved ergot to be the cause of disease. Dodard, Aitken¹ states, produced ergotism in fowls by feeding them with diseased rye in 1626.

Raw padi, or clean or refuse rice, or wild grasses affected by the assumed fungus of beri-beri, as brome and other grasses are with ergot, are possible means through which animals should become affected by beri-beri.

It is asserted that this happens :

Equine Beri-beri.—Lacerda,² in 1885, described a pestilence of horses and pigs on the Island of Marajo which he considered identical with beri-beri. The disease is termed 'hip-pestilence,' or *quebrabunda*. The affected animals, he says, show debility, uneven gait, dyspnœa, restlessness. Unable to bear support long on one limb, they shift to another. They stagger on moving. The abdomen is retracted ; constipation, dysuria occur. There is no anorexia nor thirst, rarely fever. The condition may go on to paralysis and dropsy. Ulcerations occur on parts damaged by falling about ; emaciation and atrophy ensue. The disease

¹ *Sc. and Pract. Med.*, 1864, vol. i., p. 765.

² *Berl. Klin. Woch.*, 1886.

is fatal in from a few days to months. Autopsy shows viscera swollen, blood tarry, ecchymoses in bladder and omentum, and diffuse sclerosis in the cord and nerves. Lacerda held the condition to be a polyneuritis, and attributed it to the ingestion of an 'ascomycetous' growth in water. Inoculation of this microphyte direct from the water, or cultivated, produced, he stated, closely similar¹ symptoms in rabbits, pigs, birds, and monkeys. Rebourgeon, in a paper on this same 'beri-beri' of horses, ascribes the effect to a bacterium, which had the remarkable character of being innocuous when brought to Europe, but was pathogenic in Brazil.

In India a paralytic disease of horses known as *kumri* has long been recognised, the causation of which has not been determined.

In the Malay Peninsula I have observed in several kinds of animals, but particularly in horses, a paralytic malady which agrees with the accounts of *kumri*, and which I am satisfied is due to feeding on padi.

Having lost thirteen animals from this disease, and observed many others, I have studied its clinical aspect closely.

This is what happens :

A well-fed animal in perfect condition, previously a good mover, is observed while being driven or ridden to lose 'time.' Its movements are imperfectly co-ordinated. It suddenly drags or lurches, 'gives,' or stumbles. That is often the first sign noticed of the disorder. Closer examination shows that a hind-foot drags, or both trail slightly when walking slowly. A hip is a little 'down,' or the quarters altogether are slightly dropped. There is usually tenderness over the coupling (sacro-lumbar spine), so that the beast will flinch on pressure there, sometimes even drops or crouches down nearly on to its stifles from the pain. It is unable to turn sharply, and liable to fall over if suddenly made to do so.

Or an animal perfectly well overnight is observed to have a difficulty in rising in the morning : when the same symptoms or more advanced ones will be observed. Sometimes the animal is found totally unable to rise, this being the first sign of illness noted. From this beginning paresis extends until, within a few days, or after months, distinct monoplegia, paraplegia, tetraplegia, or more rarely apparently total paralysis results. As early often as the first paresis, slight oedema may appear, first

¹ *La. Sc. Méd.*, 1890, No. 31.

about the sheath or scrotum, next in hind-legs at the coronet, spreading thence up pastern, cannon, and gaskin, up to the stifle, and sometimes all over the quarter. The lower part of the belly is usually early implicated, and the condition may extend to the chest-wall in front between the fore-legs. When œdema is advanced, the fore-legs also become anasarcous. Œdema as extensive as this is rare, but in some degree it is nearly always present. 'Capricious' patches of it, as only on the chest betwixt the fore-limbs, are sometimes seen, and may vanish as they have appeared—suddenly and without apparent cause. The tendon sheaths do not 'fill'; the joints are unaffected.

There is certainly in most cases some anæsthesia both to touch and to pain, and there seems to be itching or irritation or other paræsthesiæ, for animals will continually rub, bite, or worry certain parts upon which there is no visible change.

The disease persisting, but not ending fatally, œdema subsides, but affected muscles atrophy, and impairment of movement always remains. In bad cases there may be general atrophy of the muscles, producing great apparent emaciation, in spite of ample feeding.

The course of the complaint is variable. It may last days or years. It may subside at any stage. But usually, repeated exacerbations happen, which ultimately entail a fatal issue. There is seldom any tenderness to pressure of muscles or of nerves or anywhere but in the loin, which is often exquisitely tender, so that the animal shrinks from any touch. The general condition remains, as a rule, good. Except at the outset, the animal rarely shows any fever or other general signs of illness. There may be constipation. There is often dysuria, apparently from bladder paresis, which is overcome by straining. The appetite is unimpaired throughout.

The chief source of distress in bad cases is difficulty in getting up, or when up of long remaining so. Every effort to rise is attended with violent trembling, and the desperate exertions made lead to copious sweat and speedy exhaustion. The result is that the animal, once risen, but remembering and dreading the effort made to get up, stands still till exhausted. It then half sinks down, but through restlessness or fear of a position from which it can with difficulty stir, it immediately struggles up again, and between falling and struggling up it soon becomes worn out. Finally, quite unable to rise, it remains down, rolling about, or lies prostrate and extended, able to make only feeble

efforts to rise, or but just to raise the head. Even in this desperate strait a horse will continue to eat and drink. Death results, failing a merciful bullet, generally from exhaustion. But cardiac crises, accompanied by great distress, an intermittent pulse, and irregular heart-sounds, frequently occur, and death may happen in one of these. In several animals which I have had carefully slung and attended to the end, this has been the termination.

The disease is not necessarily progressive, but may be interrupted at any stage. If this happen early there may be complete recovery in every respect; if later, the paralytic effects and atrophy of muscle will remain.

I regret to say I have no detailed notes of autopsies made. It is usual to find an engorged right heart, and perhaps darker blood than usual, and some fluid in all the cavities. I have seen small ecchymoses in the pericardium about the omentum and in the stomach. The kidneys have appeared congested, but I have observed no marked lesions in the viscera.¹

It is obvious that the symptoms here described bear great resemblance to lathyrism, but to the form of it observed in human beings, as so graphically described by Hendley,² rather than that seen in horses, of which Leather,³ McCall,⁴ and Hayes⁵ have given accounts. The chief differences between clinical accounts of the two complaints seem to be (1) the presence of peculiar rigidity and spasticity of muscles, especially those of the back, exaggerated reflexes, clonus, etc., in lathyrism, all of which are absent in 'beri-beri'; (2) the absence of atrophy of affected muscles in lathyrism, almost invariably present in 'beri-beri.'

Although I have often carefully searched the *grain* supplied to affected animals for seeds of *lathyrus*, I have never discovered any.

In the East the food commonly given to horses consists of *padi* (rice) and *kachang* or *gram*. Black and green gram, the mung bean (*Phaseolus mungo*), catjang bean (*Vigna catjang*),

¹ I have not myself examined either cord or nerves microscopically, but in one such case which was examined by Dr. Hamilton Wright for the Veterinary Surgeon of Selangor (the latter gentleman informed me) changes resembling those of locomotor ataxy were seen. I have often sought, but never found anything abnormal in the blood.

² *British Medical Journal*, 1903, vol. ii., p. 707, and *Jour. Trop. Med.*, vol. i., p. 359.

³ *Veterinary Journal*, April, 1885.

⁴ *Veterinarian*, 1886, p. 789.

⁵ 'Veterinary Notes for Horse-Owners,' 1901.

horse gram (*Dolichos biflorus*), are all used, but the latter principally. The disease has affected exclusively, according to the writer's personal experience, those fed on padi.¹ Imported Australian, next Arab, and Indian country breeds have most often been observed to be attacked, Java, Sumatra (Delhi), Sulu, and Burmah ponies but seldom. The latter, owned and kept for hire by natives, are mostly ill-fed, and if they are fed on grain of any kind get very little of it. The former, if fed on padi, naturally have a plentiful supply. I have seen the disease in Burmah ponies owned by Europeans.

The writer, so long as his horses were fed largely on padi, lost many of them from this malady. But having gradually acquired the belief that padi was its cause he gave up the use of it, and has since, although keeping more horses, not had a case of the disease.

The following experience served to establish this conviction: In 1900, after an absence of a fortnight from home, I found two horses affected which had been left in charge of the *sais*, and exercised by walking only. The first (a mare) was paretic in both hind-legs equally, so that the front edge of both hoofs trailed on the ground at each movement forward. There was slight loin-tenderness, and general weakness, the animal swaying over and staggering on a slight push. She could walk fairly, and turn slowly without trouble. The second (a gelding) was much weaker, standing with effort, and swaying with legs widely spread out. It objected to movement, trembling violently at every effort made under stimulation, and then could walk but very slowly, all four feet, barely flexed at fetlocks, being trailed on the ground as it moved forward. It could not turn without assistance, showed puffy coronets, and had loin-tenderness. Both horses were at once given an oil purge and some bran-mashes, and put on grass. Both improved, but in the case of the gelding there was, at the end of a week, a relapse or recurrence. He grew rapidly worse, and after two or three weeks died suddenly, having been then a week in slings.

The mare completely recovered in ten days. A few weeks later I determined to experiment by giving her padi again. She at once relapsed, getting paresis in both hind-legs. This disappeared again on grass. In another month padi was tried once more, with the same result. After this no further experiments

¹ He has been assured by others that animals to which padi was not given have had the disease; but he has himself met with no such cases.

were made, and she remained perfectly well, being worked regularly. In this case there seemed to be little doubt that padi was the cause of paresis.

But the writer has been informed of similar cases in adjoining districts in animals which were stated to have had no padi, and to have been fed on 'crushed food' (a mixture supposed to contain only gram, oats, and chaff, made in Calcutta, but of doubtful composition), hay, and local fresh grass, and in India I have been informed *kumri* frequently occurs in places where neither padi nor gram, but only barley, is used.

Paralysis in Rice-fed Pig.—A pig experimentally fed on uncured rice at the Seremban Hospital, and carefully tended in every respect, being kept in a single cement-floored room, which was frequently washed out, developed paraplegia in a month, and died quite suddenly. It fed well to the last, had no fever, and no other symptoms. Nerves and cord from this animal have been submitted to the School of Tropical Medicine for examination.

Beri-beri of Monkeys.—H. Wright¹ claims to have induced polyneuritis in monkeys fed only on fruit as a result of exposure in a beri-beric 'focus.' But the conditions under which the experiment was done, and admitted septic complications in his animals, render these results valueless as evidence from the point of view of beri-beri. Septic polyneuritis is, of course, well known.

Of more account is the statement of Hose,² that monkeys under his own observation in Borneo, fed on bazaar rice, developed paresis, and that his experiments 'were repeated by the University authorities at Tokio with entirely confirmatory results.'

Hose makes a similar statement as to the production of beri-beri in fowls.

Rupert³ refers to the reputed occurrence of beri-beri in many animals, including horses, sheep, cattle, but had never himself seen any such cases. He mentions that he had seen many cases of paralysis resembling beri-beri in domesticated cats, but would not undertake to affirm they were beri-beri. Cats, it may be mentioned, in the East often enough share the rice of the house-

¹ 'Studies of the Institute of Medical Research,' vol. ii., No. 1; *Brain*, 1903-4. See also Durham's comments on these results in *Journal of Hygiene*, January, 1904, p. 128.

² *British Medical Journal*, October 28, 1905, p. 1099

³ *Loc. cit.*

hold. Ucherman¹ states that beri-beri was observed in animals on board ships with the disease. He mentions sheep, geese, and fowls as being affected.

Paralysis of Horses in Temperate Climates.—From a recent summary of work by Dechambre and Curot,² it may be gathered that forms of paralytic affection in horses, the result of mouldy or otherwise altered corn or fodder, are recognised by veterinarians. The account given is vague, and paraplegia appears to be uncommon, the troubles produced by such foods being generally of a gastro-enteritic type. An '*infectious mycotic paraplegia*' is mentioned, perhaps the *quebrabunda* described by Lacerda belongs to this class. The production of the toxic substances in the forage is referred to various moulds, ascomycetes, etc., as possible causes. It is stated that an enzootic was described by Guillemard among horses belonging to several studs, but fed on the same food at Paris, and another by Comenz among the horses of a cavalry regiment. The incriminated articles were mouldy bran, and oats affected with *penicillium*.

Ustilago maidis was taken, the same article states, by Emhoff in doses of 3 grammes daily for fourteen days without ill effects, and *ustilago segetum* in the same manner by Cordier, with no result.

The specific diseases common to animals and man are not many, and paralytic conditions of any kind in the horse especially are so rare, that the occurrence of forms of palsy clinically the same in both affords a very strong presumption of their having the same origin. In the writer's opinion the malady seen by himself in horses is beri-beri, and differs little, if at all, from the disease in man.

Avian Beri-Beri.—Rarer still are affections common to man and birds.

Yet a form of polyneuritis occurs in fowls,³ the symptoms, the course, the morbid anatomy, and the conditions of production of which agree at all points with human beri-beri.

It happened that in 1894 a singular epidemic appeared among fowls kept at the Government Laboratory at Weltevreden in

¹ *Lancet*, May 30, 1902.

² *Veterinary Journal*, March, 1904, p. 139.

³ H. Wright, indeed, has said of the polyneuritis of hens that it is 'probably not even an analogous condition' (to beri-beri). But if ascending paralysis, with internal dropsy and peripheral nerve degeneration, in any animal is not 'analogous' to beri-beri, what is the meaning of the word analogy?

Batavia, by which 41 out of 46 hens were affected, and of which 30 died. The symptoms resembled as closely as under the conditions they could do those of beri-beri in human beings. Inspection showed similar pathological changes—polyneuritis, etc. These fowls had been fed entirely on cooked white (stale uncured) rice.

Experimenting further in 1896, Eijkman¹ found that peripheral neuritis could almost invariably be produced in fowls by feeding them exclusively on decorticated 'white' rice. When fed on 'red' rice—i.e., rice still covered partly by pericarp—or whole unhusked rice (padi), this event did not follow. Moreover, affected birds recovered when 'red' rice was substituted for the white, or when husks or pericarp were added to the latter. The result was independent, it was stated, of the *source* of the rice used. In its irregular period of production, symptoms, and morbid anatomy, the disease resembled beri-beri.

Eijkman concluded that a definite poison exists commonly in rice, which produces the disease, and that for this poison or its effects something contained in the pericarp was an antidote.

Eijkman's results have been much criticised. Van Dieren² in particular shows that his interpretation of symptoms was far from definite, and his methods loose. In some cases where polyneuritis was stated to have been the cause of death the nerves were not examined; in others degeneration of the nerves being found after death, the same diagnosis was made, although during life no definite symptoms had been observed. It is not clear to the writer whether the rice used in Eijkman's cases was always *fresh* or, as seems likely, more often stale.

A perhaps very important point, assuming the possible origin of the disease from an epi- or endo-phyte, lying usually in the envelopes of the seed, or between the endosperm and pericarp, is that rice—the bare endosperms—quite freshly prepared, may be, unless freely washed before consumption, copiously contaminated with the postulated poison, thus hens would on occasion get polyneuritis even from a perfectly fresh rice. The use of the same article by men would, on the other hand, not be injurious, because washed.

Fowls are fed all over the East on unhusked rice (padi) without ill results, and this fact may be regarded as a sufficient 'control' for paralysis experimentally produced in them.

¹ *Gen. Tijdschr. v. N.-I.*, 1896, p. 214.

² *Kantteekeningen*, p. 198.

Later it was found by Grijns¹ that not only rice, but also sago, tapioca, and even *flesh*, when this had been heated for two hours in an autoclave at 120°, also produced polyneuritis. Accepting these facts, which, if confirmed, are of interest, the effect derived from rice does not thereby lose its importance, as Laoh seems to suppose. The fact that arsenic, for instance, may cause neuritis does not render the toxicity of alcohol necessarily mythical, although there be many who have, like Laoh, seemed also to hold this belief. The newer observations simply prove that more articles than rice may become the medium of the beri-beri intoxication. The more important result of Eijkman's and others' researches still remains that stale white rice entails this polyneuritis, while other sorts of rice, and the same rice diluted with other substances, do not do so. The pericarp being claimed as really antidotal to the effect of the white rice in Eijkman's experiments, it would have been interesting to learn whether it were so also for the effects of the sago, tapioca, dried meal, etc. But on this point no experiments seem to have been made.

Exception has been taken to Eijkman's results in that the experiments were conducted in an 'infected' locality; that the neuritis was induced at one time, when beri-beri generally was on the increase, and the therapeutic effects at another, when beri-beri in the district was declining.

While such objections, if sustained, might weaken the contention that the experimental beri-beri depended entirely on the rice given, as opposed to an infection, it seems to have been lost sight of that their very validity as objections depends upon accepting the fowl neuritis as being *determined by the same conditions or cause as human beri-beri*.

Sakaki, repeating Eijkman's experiments, has confirmed the observation that polyneuritis may be produced in fowls by feeding them on certain sorts of rice.

His experiments were made in Japan. The fowls, kept separate, but in contiguous coops, were under good hygienic conditions. They were given 'vegetables occasionally,' in addition to the experimental food, and allowed exercise daily. Forty-five fowls were used, being fed in groups consecutively on eighteen different materials.

Five, not fed on rice, and 6 fed on padi, or rice with some husk adhering (uncleaned rice), showed no signs of paralysis

¹ *Med. Lab. Path. Anal.*, etc., Weltevreden, 1900.

after long intervals. Of 34 fed on various kinds of cleaned (white) rice, 14 got paralysis, 20 showed none.

White Rice produced Paralysis.—Thirteen different samples were tested on the 34, in only 3 of which the results were equivocal—*i.e.*, some birds failed to get paralysis when fed on the same rice which caused it in others. Such cases may have been examples of superior resistance.

For the effect of the clean rice in producing disease, fowls not fed on rice at all, served as controls. The differences between the rices fed to affected and unaffected birds in these two groups were marked, and such as it is reasonable to suppose should have influenced the event. The conclusion that the disease was due to specific differences in the rice used is, therefore, well founded.

In all the cases in which paralysis was produced the signs were similar, and the post-mortem lesions identical. The birds refused food, showed anorexia, then staggering and inco-ordination on movement. There was weakness, and later evident paralysis, first of legs, and next of wings. These drooped heavily. Then ensued total loss of sensation of stimuli, dyspnoea, diminished respiratory frequency, and intercostal paralysis, pallor of skin, general emaciation, finally total paralysis and death. The symptoms set in usually only after many weeks.

Morbid Anatomy.—Autopsies showed œdema of tissues, and serous effusions in cavities, especially pericardium, atrophy, and cloudy degeneration of muscles, anæmia of lungs, spleen large compared with atrophied other viscera, dilated and engorged right ventricle.

In birds simply starved, signs set in earlier. There was emaciation at seventh day, they could always walk, though weak, and without staggering; the wings drooped sooner, but never in so marked a way as in paralysis. There was no dyspnoea up to the last, and increased instead of diminished respiratory frequency. At post-mortem there was little serous effusion, more atrophy, especially of liver, no right ventricular dilatation. Allowing for difference between birds and man, the parallelism between this polyneuritis of hens and human beri-beri must be admitted to be remarkably close. The broad clinical symptoms, and the macroscopic changes post-mortem, afford, perhaps, a sounder basis for the discrimination of beri-beri than do delicate differences in neurones, which can be detected only by the expert, and when described appeal least, probably, to those who know most about them. Judged in this manner, it would be difficult

to say that there is any essential difference between the polyneuritis of fowls and that of human beings, and viewed in connection with its demonstrated cause—namely, rice—it would seem that the condition described is true avian beri-beri.

Sakaki's careful observations seem so important in the light they throw on the possible influences affecting the toxicity of rice under different conditions that I append an abstract of them (see pp. 362 and 363).

What are the inferences permissible from these data?

Sakaki's own conclusions may be briefly summed up as follows:

1. 'Cleaned rice certainly contains some poison which gives rise to paralysis.'

2. Its intensity is variable, differing according to:

(a) *Sort of rice*, 'upland' (hill-padi) being more poisonous than swamp-rice (wet-padi), and 'glutinous' rice being innocuous.

(b) *Age*, rice of recent harvest being more, old padi kept for seven to twelve years being less or not at all, poisonous.

(c) *Exposure* of rice after cleaning—i.e., separating pericarp—rice used immediately after cleaning—fresh rice—being innocuous, that exposed for ten days or more—stale rice—becoming toxic.

(d) *Specific qualities*, derived from different manures during culture, rice treated with lime being most, with human faeces next, and with fish manure least, or not at all poisonous.

3. In rice freshly cleaned and sterilized by dry heat (150° C.), and kept so, no poison appears. '*It is produced by micro-organisms which invade it from the air.*'

4. Rice already become toxic is unaffected by steaming (100° C. half to one hour).

5. 'Leaving out the question whether this rice is the cause of kakké or not, it is quite evident that it must be poisonous for human beings, since it certainly is so for animals.'¹

Sakaki's experiments certainly seem to show that *cleaned ('white') rice sometimes contains a poison which causes paralysis*, and that as Eijkman affirmed, the same rice which with its envelopes on (husk and pericarp, or part of the pericarp only, padi, or 'red' rice) is innocuous, may become toxic after it has been cleaned. But some other of his conclusions the evidence is far too meagre to support, and the facts even may be interpreted otherwise. In particular, his explanation of the origin of the poison in cleaned rice as being the result only of germs

¹ *Sei-i-Kwai*, March 31, April 30, 1903; see also abstract in *Caducée*, 1903, vol. iii., p. 278.

invading it from the air—in fact, *decomposition*—is open to objections, several of which the experiments themselves afford.

The view implies that raw (uncleaned) rice padi itself contains no poison, that cleaned 'white' rice freshly used as prepared daily from the padi—in fact, 'fresh rice'—is not toxic, because the air-borne germs (saprophytes) have not yet had time to invade and render it poisonous; but that if sufficiently exposed, for ten days or more—*i.e.*, when the clean rice is *stale*—this will happen.

Grounds for Saprophyte Theory.—The main ground for this contention is, of course, the experiment (*l*), in which a rice freshly prepared, and of a sample which (it is left to be inferred, for this is not definitely stated in the text) had been proved by other tests to be toxic, when put in a bottle and dry-sterilized, and kept from contact with the air, remained harmless, given to two fowls for a long period. But seeing that in other experiments—(*h*), (*m*), (*n*), (*o*)—the result was equivocal the same rice which affected some birds proving harmless to others, it might well be supposed that the two birds in experiment (*l*) also escaped paralysis through their own powers of resistance, as seems to have been certainly the case with one bird in (*o*).

Only seven birds in all were fed on 'fresh' rice (*g*), (*h*), (*l*); one got paralysis (*h*). The rice used for *three* of them (experiment *g*) was of a sample which, in another experiment (*a*) used when stale, produced paralysis in two birds, yet failed to affect a third (*a*). In experiment (*g*) none of the birds was affected. In other *two* birds (experiment *l*, that of the dry sterilized grain) the exact source of the fresh rice used is not stated; and it might well have been harmless, even had it been used stale, as was the case with the stale rice from the fish manure (*m*).

In another *pair* of birds (experiment *h*), although the rice used was the same (seven year old padi) which another experiment (*j*) showed to be at times harmless, even when stale; one of the two birds fed on it *fresh* got paralysis.

Moreover, it is difficult, on the decomposition theory, to understand how, if the poison is produced in the rice only after cleaning, such influences as species, age, locality of growth (upland or swamp), or manuring during culture, should have the remarkable effect which Sakaki claims for them.

To explain this it would have further to be assumed that the saprophytes require for their growth, and the elaboration of the poison, some special substance, the original production of which in the grain may be wholly checked by many slight and different factors. As a speculation this is possible; but it does not seem probable.

362 THE CAUSE AND PREVENTION OF BERI-BERI

RICE AND POLYNEURITIS IN FOWLS: ANALYSIS OF SAKAKI'S EXPERIMENTS.

Number of Experiment.	Character of Rice used.	Numbers of Fowls.	Result.	Remarks.
I.	Last harvest padi	1, 2	Paralysis	One died at 120, 1 at 124 days.
(a)	Ditto, cleaned and exposed 10 days	3	No paralysis	Three killed accidentally at 138th day.
(b)	Ditto, uncleaned	4, 5	No paralysis	Both healthy after 268 days.
(c)	Unhulled padi	6, 7, 8, 9	No paralysis	Both healthy after 268 days.
Control	On cracked barley	10	No paralysis	But died suddenly of pneumonia.
II. (d)	Upland padi, last harvest, cleaned and exposed 10 days	11, 12	Total paralysis	Of which both died at 24 and 25 days.
(e)	Twelve-year old padi, cleaned and exposed for 50 days	13, 14	No paralysis	One died 51st day of pneumonia, one 204th day of uncertain cause, but certain no signs of paralysis.
(f)	'Glutinous' padi, last harvest, cleaned and exposed over 10 days	15, 16	No paralysis	Healthy after 337 days.
III. (g)	Last harvest, fresh-cleaned daily	17, 18, 19	No paralysis	Healthy after 250 days; 19 died suddenly 137th day; had ulcer of stomach.
(h)	Seven-year old padi, same sort as (e), fresh-cleaned daily	20 { 21 {	General paralysis No paralysis	} Died 157 days. } Healthy after 220 days.
(i)	Same as (h), but exposed 10 days	22, 23	No paralysis	Healthy after 220 days.
IV. (k)	Upland padi, cleaned and exposed 10 days, and sterilized by boiling	24, 25	Paralysis	Both died at 20th and 21st days; sterilized by boiling at 100° C. half hour to one hour daily as used.

RICE AND POLYNEURITIS IN FOWLS—(continued).

Number of Experiment.	Character of Rice used.	Number of Fowls.	Result.	Remarks.
IV. (l)	Fresh rice, dry-sterilized as cleaned, and kept sterile	26, 27	No paralysis	Healthy after 142 days. This rice was sterilized by dry heat at 150° C., and kept in sterile airtight bottle.
V. (m)	Grown and manured experimentally with fish manure, cleaned and exposed 34 days	28	Paralysis	Died with severe gastric catarrh 62nd day; before death showed 'slight paralysis.'
	Ditto	29, 30, 31	No paralysis	Healthy, and increasing in weight after 266 days.
(n)	Grown and manured experimentally with human faeces, cleaned and exposed 34 days	32, 33	Paralysis	These died with general paralysis at 49th and 69th days.
	Ditto	34, 35	No paralysis	Healthy after 266 days, and gaining weight.
o)	Grown and manured experimentally with lime, cleaned and exposed 34 days	36, 37, 38	Paralysis	All but one died. Paralysis began in 36 at 11 days, died 23rd; in 37 began 29 days, died 49 days; 38 died at 62 days.
	Ditto	39	No paralysis	Thirty - nine showed wasting 66-70 days, recovered, and was well at 366 days.
VI. (p)	Upland padi, cleaned and exposed over 10 days	40, 41	Paralysis	Died at 20 and 91 days.
Controls (q)	Controls given only water	42, 43	No paralysis	Began to emaciate 7th day; wings drooped before legs weakened; no staggering, dyspnoea, or anaesthesia.
Controls (r)	Varied food	44, 45	No paralysis	Remained healthy.

The balance of evidence which his own experiments afford seems, then, to be against Sakaki's theory of origin of the poison. The fact is none the less to be accepted as demonstrated that exposed clean rice becomes poisonous which was not so when fresh. It may even be taken as proved that the effect of sterilizing the rice in experiment (*l*) was to prevent the development of poison in it, and so the inference made probable that the same process will usually have this result. But both results may depend on other conditions; the facts are susceptible, it will be seen later, of other explanations.

The differences observed in the poisonousness of different rices according to their *age* after harvesting, their *source*, and mode of *culture*, seem better established. Thus, taking the experiments only in which rice cleaned and exposed—*i.e.*, *stale white rice*—was used, and excluding 'glutinous' rice, which is practically a different species (*Oryza pūlut*), then we have nine occasions on which twenty-five fowls were fed with different samples of such rice—(*a*), (*d*), (*e*), (*j*), (*k*), (*m*), (*n*), (*o*), (*p*). On six occasions paralysis in thirteen birds . . . was produced. 'Upland' rice (hill-padi) proved poisonous on all the three occasions, and to *all* the birds (six) on which it was tried—(*d*), (*k*), (*p*). Swamp-grown rice was used on the other (six) occasions, and on three proved harmless to *all* the eight birds to which it was given—(*e*), (*j*), (*m*). Of nineteen birds altogether fed on swamp-rice, paralysis was produced in only seven. The samples used included two of *very old stock*, seven-year old padi (*j*), and twelve-year old (*e*), which were both innocuous. Excluding these two, we still have swamp-rice less toxic than the hill-padi, being entirely harmless to four birds tested on one occasion (*m*) out of four (unless to twenty-eight 'slight paresis' be reckoned) occasions, and to some only of all the birds tested on the three other occasions; or, altogether, to eight birds out of fifteen tested, stale swamp-rice not aged proved harmless.

The different results obtained with the same sort of rice grown experimentally at the same time and in the same place—the Government Gardens—but manured with different manures, are remarkable. In ascribing the irregularity of results obtained with them to *the manure* used in culture Sakaki has, as pointed out, adopted a view almost irreconcilable with the explanation of the development of the poison in the rice subsequently to cleaning on the advent of germs.

Differences in *vigour* of growth, dependent on whatever factor,

are, however, well known to influence the liability of plants of all kinds to pests, blights, and fungi, and Sakaki's results seem rather to indicate that in some such a secondary phenomenon as this, most likely, the true explanation lay of the observed differences both in his experimentally-grown rices and in hill-padi, as distinct from wet-padi.

It often happens that an exception is more important than the rule from which it departs, and of this experiment (*h*) seems to be an example worth emphasis for more reasons than one.

In (*h*) two birds were fed on fresh rice made from the same cleaned seven-year-old padi, which in (*j*) proved harmless, even after exposure for more than ten days after cleaning. Yet, although the rice was used as freshly cleaned daily, one of the birds fed on it got general paralysis at 157 days.

This is *proof that the poison does not always require exposure after cleaning for its development*. In some cases, at least, therefore, since it is to be found in freshly-cleaned rice, it must be presumed it will be present also in *padi* before husking. This accords with the fact earlier noted that unhusked padi produces paralysis in horses.

Petit¹ offers, in support of Eijkman's views, some results obtained by himself in Madagascar.

'During 1898,' he says, 'the Senegal Tirailleurs in cantonment on the West Coast of Madagascar, on the Tsiribihina, and at other stations, were attacked by beri-beri, which occasioned a pretty severe death-rate. Prophylactic and hygienic measures prescribed gave but poor results, owing to the difficulty of applying them in a district in active insurrection.

'In 1899 conditions had improved somewhat, but nevertheless between February and July 250 per 1,000 of the active strength of the Rifles were affected by beri-beri. The rice distributed to the men at this time was bad in quality, so I set myself to repeat Eijkman's experiments, who suggests that the rice-envelope contains certain principles capable of neutralizing the poison which engenders beri-beri. I had 300 grammes (10 ounces) of padi given to the riflemen in place of 100 grammes (3½ ounces) of rice. At first they demurred somewhat, saying "padi was made for mules, not for them." But they agreed, however, to have the padi half peeled, cooked, and mixed with the rice, when they had found that many of their comrades removed from

¹ *Ann. d'Hyg. et de Méd. Col.*, 1903, vi., 1, p. 98.

posts close by were beri-beric, while (*sic*) they remained exempt.¹

'Padi has been used at a great many stations, and has given good results. The men took every morning a large glass of padi decoction, the diuretic properties of which soon became apparent in the disappearance of cedema in mild cases. I have pushed the dose to 2 litres of decoction daily. At all the stations where padi was used it gave good results when the patient had been not more than a few days ill, and in cases of slow course. In cases of rapid type, if from the first day of using the decoction there had been no increase in the urine, this not exceeding 100 grammes, there was no hope at all of saving the patient.

'It results from my observations that padi mixed with rice and decoction of padi certainly have a neutralizing effect on the poisons produced in bad rice.

*'There has never been observed a single case of beri-beri among the natives living in villages near the stations, and this is due, I believe, to the fact that they store their rice as padi, which they husk daily for the day's requirement.'*²

'Some observers have supposed that beri-beri was due to eating salt fish, to want of nitrogen, or of fat in the dietary. This is not true.

'The rations of the riflemen at some stations never included any salt fish, and at others they had, in addition to an allowance of 800 grammes (26½ ounces) of rice, 500 grammes of fresh meat nearly every day. Moreover, they got often fresh fish for themselves, poultry, oil, sugar, etc.

'But notwithstanding all these adjuncts, which they were able to get over and above their rations, none the less some cases of beri-beri appeared.

'I have even noted two cases in soldiers who were under treatment in the ambulance, where they were comfortably lodged and fed, but it is true that their staple food was Government rice, the only sort just then obtainable.

'There is reason, then, to believe that the latter is what must be incriminated, and I consider that Government would show great wisdom if, in order to prevent the recurrence of such mishaps, they firmly proceeded to have part of the rice-rations made to consist of padi.'

The facts recorded by Petit thus bring from quite a new quarter

¹ From which it would appear that these persons were already distinguished by 'immunity' before they agreed to the diet of husks.

² Present writer's italics.

of the world valuable confirmation of some of the views already put forward in this paper. The production of beri-beri among the Senegalese troops on service in Madagascar clearly depended on excessive use ($26\frac{1}{2}$ ounces) of stale white rice, and the result to them is contrasted with the immunity of natives of the country using only fresh rice.

But the observations on which Petit relies as confirmation of Eijkman's views are capable of another interpretation.

Where 100 grammes of bad rice were replaced by 300 grammes of padi (fresh rice), *the total quantity of stale rice consumed was* thereby first *reduced* absolutely by 100 grammes, and the relative effect of this reduced quantity rendered still less by dilution with other 200 grammes of padi (or fresh rice). Such a proceeding alone, quite apart from any antidotal properties in the husks which were added, would be quite sufficient, all the evidence adduced previously goes to show, to determine the disappearance of beri-beri.

In the cases when padi decoction was employed, there is no discussing the conclusions, as sufficient details are not given. It is obvious that the mere fact of amelioration of symptoms in some mild cases affords nothing more than a presumption that the decoction may have been of value in promoting recovery. But such cases occur constantly without use of any remedy. On the other hand, the severe cases, which ought to have responded, at least in some degree, to a real antidote, showed no impression.

The production of beri-beri through rice in animals is important, not so much as additional proof of the dependence of the disease on that cause in man—for this the writer believes sufficient has already been adduced to demonstrate—but as throwing light upon some of the conditions under which the poison in the rice is produced.

In the writer's view the connection between human beri-beri and rice having been first and independently proved, the clear dependence of the paralytic disease of hens and horses upon rice also, in some form or other, is the proof that their malady is really beri-beri.

But for those who do not appreciate the evidence brought forward as proving human beri-beri to be caused through rice, the production of a form of paralysis clinically undistinguishable in men and animals, under conditions in which rice is the only demonstrated common factor, must afford a strong presumption that the condition has the same nature and origin in both.

SECTION VII

NATURE OF THE TOXIC AGENT IN RICE WHICH PRODUCES BERI-BERI

It has been proved that beri-beri is caused by the consumption of stale white rice—the bare seeds stripped of all their envelopes, as well as the aleurone layer which forms their surface.

But the exact nature of the process by which this result is brought about remains obscure.

While it seems reasonable to conclude that the disease is but a simple intoxication due to the absorption of some positively poisonous substance actively present in the rice, according to the rigorously logical method which it has been sought to follow in this paper, it may be urged that no more has been proved than that the disease follows the continued consumption of such rice, but not that the latter contains any positive toxic body.

The disorder, although undoubtedly due to the use of the rice, it may be argued, is induced not because the latter contains an active poison, but because it is actually lacking in something usual to or normally present in it ; or, where articles other than rice constitute the diet, in those articles.

In this, a new form of the dietetic or physiological view,¹ and which may be called the *privatory* theory of origin of beri-beri, such main components of diet as fat, proteid (or nitrogen), vegetables, salts, etc., are not meant to be included. The evidence by which the various 'scorbutic' views have been disposed of, and the argument which in especial applies to them—namely, that beri-beri only appears irregularly in communities whose diet is nevertheless for long periods continuously the same—does not apply to it. The quality now postulated as

¹ I owe it to Professor J. S. Haldane, who very kindly criticised this work before it went to press, that the possibilities of this view were made clear to me.

lacking in food—of whatever sort : rice, wheat, corn, sago, tapioca, even flesh—the consumption of which entails beri-beri, is nothing coarsely recognisable, nothing determinable by chemical analysis, or indeed by any test other than the effects which deprivation of it may produce. It may be something necessary to the normal digestion or assimilation of carbohydrates only, or of all classes of food.

To account for the irregularity of production of its effects—the periodicity of the disease—it would be supposed that the principle was periodically absent, more or less entirely, from the food-stuffs concerned, which would in fact, without it, be themselves in a sense the actual toxic agents.

In support of this view there are certainly the facts (1) that beri-beri occurs most often under circumstances a common factor in which is complete or partial privation of some component or another of a good dietary, or general depravation of them all ; and (2) that epidemics are checked, and also the course of the disease in individuals, when certain articles, such as may be supposed to restore the lacking element, are added to the rations—often in quite small amount. Fat, fresh meat, fresh vegetables, the pericarp of rice, appear to be such examples.

The fact that beri-beri does not follow the use of fresh, or cured, or red rice, and rarely (if indeed ever) the use of other staples than rice, but is induced only by eating stale uncured rice, would be explained, on this privatory theory, by supposing that : (a) fresh rice always contained the necessary principle, but that (b) in stale uncured rice it more or less rapidly disappeared through the physical, atmospheric, or other changes attending its exposure, and that (c) in cured rice it was permanently preserved, fixed, or prevented from becoming destroyed, either as a result of the boiling, or in consequence of some protective action of the retained layer of aleurone cells which characterizes cured rice.

The view would receive further support from Eijkman's statement that the addition of pericarp to otherwise toxic white rice prevents the polyneuritis which otherwise ensues in fowls fed exclusively on the latter product, and from the experiments of Grijns, who is stated to have produced the same sort of polyneuritis by feeding fowls exclusively on either sago, tapioca, or even dried, overcooked, and sterilized flesh. But, apart from the difficulty of conceiving that white rice and many other articles of food thus not only normally share, but also are liable periodically

to *lack* a given principle, without which they are practically rendered poisonous when consumed, the objection seems to the writer insuperable against the privatory theory, that *fresh rice is never toxic*. It never produces beri-beri, in however large a quantity consumed, and with however little of other adjuncts it may be taken. But to explain the irregularity of occurrence of beri-beri in communities whose diets were continuously the same it had to be asumed that some quality in their food was periodically lacking. The fact that in fresh rice the quality is never lacking seems a proof therefore that it cannot be to any such periodic absence of any principle from their food that its subjects acquire beri-beri.

Failing any other alternative, then, is not the conclusion justified, that it is to the action of a poisonous substance positively present in stale rice (and other articles) that beri-beri is due?

Summary of Points proved.—A summary of the principal points so far established will enable the conditions under which poison is produced in rice to be more definitely stated, and perhaps some approach to be made to a realization of the nature of the actual agent at work.

1. Stale Decorticated Rice causes Beri-beri.—All over the world beri-beri occurs, and (with rare and doubtful exceptions) only occurs among those who eat rice which has been decorticated (white rice), and thereafter exposed under conditions which render it not merely old (as rice), but decayed or stale. The constant, the only common factor in the circumstances of those who, in thousands of instances, and under every variation of race and occupation, of locality and climate, thus got beri-beri, is the eating of such rice. Moreover, when, in the presence of beri-beri, the use of the rice is abandoned, or even the quantity consumed diminished, the disease disappears. Experiments on animals are attended by like results.

First Conclusion.—*Stale decorticated (white) rice, therefore, at times contains a poison, the effect of which is to produce beri-beri.*

2. Source of Stale Rice Variable—Toxic Effects Constant.—Beri-beri may, and commonly does, follow the use of stale decorticated, uncured rice, whatever the region—India, Annam, China, Japan, Malaya, America, Madagascar—in which the padi may have been grown, whatever the conditions under which it may have been reaped or milled or stored. But this effect does not, so far as is known, ensue from the use of any other cereal.

The degree, the intensity of toxicity of such rice, may vary, and undoubtedly does so, with numerous external topical factors which promote or retard its formation in the grain, which hinder or favour its activity when consumed. But in their essential nature the effects produced by the poison, within a clinically limited cycle of variance, are always the same ; the symptoms are constant.

It is not conceivable that a result thus unvarying could be produced by different agencies, nor is it probable that any single agent capable of producing such a uniform result—viz., the formation of the same toxic body in a host of different samples of grain—should always be found under the multitude of different conditions in which the poison is actually formed. In other words, the development of the poison in rice cannot be ascribed to any *casual* agent, such as the ubiquitous bacteria which determine common decomposition, but must be attributed to something specially, intimately associated with this particular species of grain.

Second Conclusion.—The agent which produces the poison in rice is specific of, or peculiar to, that grain.

3. Fresh Decorticated Rice does not cause Beri-beri.—Those who eat only fresh rice—i.e., the grain newly stripped of its envelopes (husk and pericarp), and not thereafter exposed for any long period—escape beri-beri. This is the experience, constant through generations, of hundreds of thousands of natives who throughout Malaya, where the disease is more than anywhere rife, and in Siam, Burmah, China, Madagascar, where it freely occurs, remain uniformly free from beri-beri so long as this habit is observed, but succumb to the malady when it is neglected.

In this immense body of evidence we have definite proof of the fact—otherwise never doubted—that rice itself, *qua* rice, the bare stripped seeds of padi, is entirely harmless. In other words, it may be formally stated, as a

Third Conclusion.—The beri-beri poison is not preformed (or not present in quantity sufficient to cause symptoms) in normal fresh rice-seeds, but is adventitious.

4. Partially Decorticated (Red) Rice rarely causes Beri-beri.—This was one of the remarkable results obtained by Vorderman from his research in Java gaols.

It will be remembered that in fifty-one prisons where stripped or scoured white rice was eaten, among an average total strength

of 9,496 prisoners there were 4,201 cases of beri-beri ; while in other thirty-seven prisons fed on unstripped or red rice (or half-peeled rice, as Von Dieren calls it), with an average strength of 6,295 prisoners, the cases were only 9 ! (see p. 205).

These 9 all happened in one prison, and all at such varying but long periods after incarceration that it could not be doubted that the disease arose in the gaol.

The reason for the extraordinary exemption of those fed on red rice, it may be said, in anticipation of a later paragraph, appears to have been due, not to any positive action of the pericarp left upon the partly decorticated seeds, but probably merely to the fact that, whether as a consequence of its presence or for other reasons, such rice was *fresh*. Here only the apparently meagre deduction (the importance of which may be seen later) is made from the facts, that in red rice, as commonly met with, there is little or no beri-beri poison, or

Fourth Conclusion.—The pericarp of rice, like the seeds when fresh, contains little or no poison.

5. Poison arises only after Decortication.—We have therefore so far evidence that a poison appears in rice some time after decortication which was not present in it (either in the seeds or their pericarps) in toxic quantity when fresh.

The material, the rice, becomes changed in its actual chemical composition by some process which begins only, or with the rarest exceptions only, after the removal of their envelopes from the seeds.

The age of the seeds, the padi, before decortication, would appear to be of no influence in this respect, since fresh and correspondingly innocuous rice is prepared and eaten by natives all over the world from padi of every age ; while, as Sakaki's experiments show, decorticated rice becomes toxic after exposure, even when made from padi seven years old, though twelve-year-old grain failed to become so.

The change follows decortication ; but that process is not alone its cause, nor, possibly, even a necessary factor in the issue. Mere ablation of the envelopes of seeds cannot, it is obvious, effect chemical change in what remains of them. It is merely that their removal permits some other influence to become operative which was impossible so long as they were intact. It may be for this reason alone that red rice remains usually innocuous, the presence on the grains of such rice of part or all of the pericarp being, for the purpose of preventing the toxic alteration, as effective, or

nearly so, as all the envelopes. The only body of evidence available—Vorderman's experiments—is not detailed enough to enable this point to be determined; but in one of the gaols under his observation, it will be remembered—that of Bangkalan—cases of beri-beri appeared, although the rice used was unstripped, or red. This, of course, disposes of any contention that the pericarp itself is of preventive or antidotal effect. It also affords the inference that the presence of pericarp on rice will not always check the development in it of the beri-beri poison. The event was susceptible, again, of the possible explanation that such white (stripped) grains as were mixed with the unstripped (red) ones in the rice used at Bangkalan—the record shows that there was a proportion of them—were sufficiently toxic to induce beri-beri even when diluted as largely as they must have been with the non-toxic red grains. Again, beri-beri, Vorderman states,¹ was known to be prevalent at Bangkalan among the villagers, who ate, as usual, freshly prepared rice. In this case fresh rice itself would have to be accepted as being—as there is no reason to doubt it must be—occasionally also toxic.

But the broad fact remaining, that ablation of the envelopes, though itself incapable of inducing toxic change, is nevertheless almost invariably followed by, or rather is a necessary precursor of, such change, has to be explained; and the necessary, and at the same time sufficient and simple, explanation would appear to lie in the fact that decortication of the seeds entails also their devitalization.

To strip from any seed its natural envelopes is to imperil, and sooner or later to destroy, its germinating power. Padi from which husk and pericarp, or even the former alone, has been, however gently, removed, rapidly perishes. Planted in soil in the ordinary way it fails to come up; when, if treated carefully in a germinating chamber, it begins to sprout, the growth soon aborts. Now, in ordinary processes of hulling both the envelopes are removed, and the seeds are at the same time exposed to mechanical violence, which kills the embryo. The seeds (the rice) are then nothing more than so much inert organic matter, liable to any form of decomposition—a suitable medium for the growth of any saprophyte.

Their devitalization, again, does not of itself alter, any more than decortication does, the chemical constitution of the seeds. But since this alteration does occur, the rice being made poisonous

¹ 'Onderzoek,' p. 62.

thereby ; and since the change which takes place, the poison which is formed, is both peculiar to this grain, and in its results or effects is constant—in short, because the product is specific—it must be inferred that the poison is the product in the rice of some agent—some ferment or saprophyte—for which decortication and devitalization merely afford the necessary opportunities of infection, and prepare a suitable medium.

Fifth Conclusion.—*The formation of poison in stale rice is due to the action of a specific agent upon the dead seeds.*

6. Decorticated Rice made from Boiled Padi is Innocuous.—

White rice made from padi, which has been boiled in the husk and then dried before stripping off the envelopes—to which the writer has applied the term ‘cured’ rice—is always innocuous at whatever period after cleaning it may be eaten, whether, in fact, it be stale or not.

Not only so, but such rice once cured may be, and often is, owing to faulty curing, especially insufficient drying, grossly contaminated with common bacteria and moulds of many kinds—in short, is to be found often much disintegrated and decomposed—and is consumed in this state without ever entailing beri-beri, although a variety of disorders of a dyspeptic nature may be due to it.

The evidence for this has been given. It rests on the experience of thousands of Tamil coolies, who, though dwelling for a continuous series of years in a region throughout which beri-beri has been as continuously endemic, even at centres where it has been pestilentially epidemic, and living fellow to others who have acquired the disease beside them, have remained uniformly exempt from the malady, while using for their food only cured rice, but have succumbed equally with others when dieted on rice of other descriptions.

This fact—of capital importance among all the data gained—seems susceptible of but two possible explanations: (1) Either cured rice is rendered by the process of curing incapable of serving as a medium for the formation of the specific beri-beri poison, or, in other words, for the growth of the agent which secretes it ; or (2) the specific agent which forms the poison is itself destroyed by the curing process.

It has been pointed out earlier in the work that grains of rice which have been cured differ in several respects from sorts not so dressed. The pericarp is as a result of the maceration made to separate more easily from the seed, and the surface of the latter,

the layer of cells in which nearly all the proteid is contained in the aleurone grains, is little disturbed. The rice is thus far more nutritious, the surface of the seeds, left smoother and less broken, may be conceived to be better mechanically protected, and the whole grain is as a rule rendered less brittle, compacter, and more tough.

Can these physical changes be supposed capable of preventing the growth in cured grain of any noxa which would cause the uncured variety to decay ?

A simple experiment will convince anyone to the contrary. If some seeds of cured and uncured rice be placed beside each other with some sterilized water in a Petri dish, the lapse of a few days will reveal an extensive and prolific flora arising equally and indifferently from or upon both sorts of seeds. Scrapings from the interior of the grains of either kind sown on plates or in broth yield equally various crops of common bacilli and bacteria. Indeed, owing no doubt to the moist condition in which locally prepared 'boiled' rice is usually put on the market, growths are generally found to be more numerous among cured than uncured sorts of grain.

Can the chemical composition of the rice be supposed to be altered by the boiling so that it will no longer serve for the beri-beri decomposition ?

This seems unlikely. Salts will be removed and proteids coagulated, but the starch, the bulk of the seed, is left unchanged by boiling. But this is hardly a chemical alteration such as could be supposed likely to entirely abolish the capacity of the boiled material to be a medium for a growth for which, when unboiled, it was fit. Moreover, the fact just noted that, considered as a medium for so many other growths (moulds and bacteria), no differences are revealed between cured and uncured rice, renders it very improbable that the reason for the non-development of the beri-beri agent in cured rice lies in any alteration of its chemical nature.

On the contrary, seeing that no change of importance can be effected in the composition of rice-seeds by boiling, it must be believed that boiled rice-seeds are just as suitable as any other for the production of the poison of beri-beri, *when the agent which induces that change is added to them*, and the conditions are otherwise suitable.

Such a point must be left to future observation to establish ; the *possibility* of the happening need only be noted here.

But of the fact, invariable in experience, that in cured rice the beri-beric decomposition does not occur, explanation is simply enough found in the second of the alternatives named above.

All agents which induce decomposition, whether in common or specific forms, whether they be ferments native to the material which decays, or micro-organisms secreting such ferments, are destructible by boiling. Since boiling destroys the capacity of rice to undergo the specific beri-beric change, therefore it must be inferred that the first factor upon which the formation of beri-beri poison in rice depends is the presence in raw padi, or in rice freshly made from such padi, of some ferment or living organism, which boiling destroys, and, as a corollary, since rice so treated never subsequently becomes (so far as is known) affected by the beri-beric change, that this toxic agent is not found elsewhere than in padi, or rice made from it—is, in other words, a specific ferment, or some parasite or epiphyte peculiar to that grain.

Sixth Conclusion.—*The poison of stale rice has an antecedent in fresh rice. The agent must be, therefore, some ferment or parasite or epiphyte peculiar to padi.*

Sum of Positive Results.—The position so far established, then, is that the beri-beri poison is a body formed in rice when stale under the influence of an agent originally present in, and, so far as is known, peculiar to the grain. This agent may be either a ferment or an organized body, and it is destroyed by boiling. These conclusions rest on a broad basis of observed facts, the evidential validity of which cannot be gainsaid.

Any attempt to determine more closely what chemically the poison, what biologically the process, what morphologically the agent, involved in the whole happening may be, must necessarily be, speculative.

The questions which remain to be answered are :

- (1) What exactly is the poison which toxic stale rice contains? What are its chemical properties and relationships?
- (2) What is the process, and of what sort is the agent, upon which its production in rice depends?

Is the latter merely a *ferment* or enzyme native to the seed, the process, a form of simple fermentation, of which the results are specific because the agent or the material is so?

Is the agent (the prime beri-beric noxa) an organized body producing the poison through similar fermentative action, the result of enzymes secreted into the rice medium by itself, or

an organism growing among the seeds without fermentation, though at their expense itself poisonous?

If organized, does the agent resemble those other organisms—moulds, cocci, bacteria—which can be spread abroad by wind and water, and so are infectible from one plant to another? Or is it some non-efflorescing fungus of those rarer sorts which are symbiotic and only propagated with the propagation of the plants, their hosts?

In the present state of our knowledge definite answers can be given to none of these questions. But there are certain general observations, some particular experiences, consideration of which may throw light upon the direction in which research may be most fruitfully made, and so perhaps ultimately aid in the solution of the problems.

7. The Nature of the Poison—Physical Properties.—The culinary conditions to which rice is submitted, which the after-results of its consumption prove to be toxic, permit certain inferences to be made as to some physical properties of the poison. Thus by most of its consumers rice is, before cooking, very copiously and thoroughly washed with cold water.

Indians, Malays, and Japanese are especially careful to do this, Chinese less so, and it has seemed to the writer that there may be some relation between the extent of this habit of washing their rice and the incidence of beri-beri on those who consume it. In gaols and other public institutions the rice is often steamed in a closed vessel to cook it, and is perhaps not previously sufficiently well washed. Were the poison a matter soluble in cold water it would thus get more or less washed away, so far as it lay upon the surface of the seeds. But the uncooked grains absorb no water, and any poison in the interior of them could not be so removed. Now, beri-beri undoubtedly has appeared often—the writer has himself seen cases—in persons whose rice has been always most scrupulously cleansed, hence the inferences gained from the facts must be:

(1) That *probably* the poison is little if at all soluble in water (cold); and

(2) That, whether soluble or no, a part (probably most) of the poison is contained within the bodies of the seeds.

Again, since all rice is at least thoroughly boiled before eating, it is clear that the poison is *not volatile*. Nor is it dissociated by a considerably higher temperature, since H. Wright has stated that the inmates of Pudoh Gaol continued to get beri-beri when fed

on rice which had been steamed under two atmospheres of pressure.¹

Toxicological Affinities of the Poison.—If beri-beri be regarded, not in its later stages, when the conditions are those due to wrecked neurones and dead or dying axons and nerve-ends, but at the beginning and in acute cases, when the symptoms of reaction to the poison are more varied and distinguishable, it will be seen that the conditions to which it presents most resemblance are those of intoxication by certain alkaloids, and more especially the condition known as *ptomaine-poisoning*.

First may be pointed out many effects comparable to those of the vegetable alkaloids :

1. In the first or early stages of intoxication—*i.e.*, under the action of small doses—there is stimulation of motor nerve-endings in the muscles (as with morphine, nicotine, pilocarpine), perhaps of the muscle fibres themselves, and excitation of the spinal motor cells (morphine, strychnine, thebaine). These effects are revealed in the increased response of the muscles to mechanical stimuli, such as light tapping, which in all early beri-berics produces immediate contraction (hypermyotonus), and in the exaggeration of the various tendon reflexes.

2. A little later (larger, *accumulated* doses?) there is depression of vagus cardiac centre (aconitine, veratrine, atropine), or perhaps stimulation of the accelerating centre (caffeine, delphinine), and depression of the vagus respiratory centre. These actions appear in the increased pulse-rate and respiratory frequency, especially on slight exertion. About this stage, too, if not at the earliest, there is general depression and languor, due perhaps to depression of sensory nerve-endings (as with nicotine, veratrine), or of brain (atropine, hyoscyamine, caffeine, cocaine, morphine), or perhaps to both combined. There is also undoubted diminution of muscular power of work, such as emetine, cocaine, quinine, apomorphine induce. The vomiting centre is stimulated (early nausea, and in grave cases severe vomiting), as with apomorphine and emetine.

3. In still later stages there is depression of most sensory nerve-endings, anæsthesia, paræsthesiæ being produced (compare aconitine, veratrine, atropine, morphine, cocaine). The vagus, cardiac, and respiratory centres have become over-irritated, depressed, destroyed (tachycardia, allorhythmia, respiratory paralysis); the motor nerve-endings are largely destroyed (compare conine, atropine, sparteine, and cocaine); many neurones, both motor and sensory, in the spinal cord are also gone (compare alcohol, chloroform, cocaine, eserine, veratrine); and there are very grave disturbances, due to depression of vasomotor nerves or their centres (vasomotor depressants, atropine, nicotine, emetine, aconitine, veratrine,

¹ Wright makes a great point of this, as proving in some way that such rice cannot contain a toxin. I find no authority for his belief that all toxins are destroyed so. But since it is unnecessary to suppose the rice poison to be a toxin, in the limited sense of a bacterial intracellular product, so it is perhaps idle to refute Wright's views as to their properties.

lobeline). Very like vegetable alkaloids in their toxic actions on the system are the bodies described as 'cadaveric alkaloids,' or ptomaines, and toxalbumins, to some one or another of which, or to mixtures of them, are attributable serious and often fatal effects in cases of meat- or sausage-poisoning.

Both these classes of bodies are due to the action of bacteria upon organic matter, which they cause to putrefy or decompose.

The *ptomaines* are crystallizable bases containing nitrogen, in chemical constitution closely resembling the vegetable alkaloids. Some are toxic, some not.

Associated with them often, but usually formed earlier in the process of decay, are less stable and greatly more poisonous bodies, named *toxalbumoses* or *toxalbumins*.

'These are,' says Mann,¹ 'proteid substances which have been acted on by bacteria in such a way as to develop intensely poisonous qualities. Toxalbumoses are unstable, and their constitution is but little understood. . . . All these substances—toxalbumoses and ptomaines—are liable to lose their toxic properties as decomposition advances. . . . Poisonous bacterial products, though unstable, are sometimes very resistant of a high temperature—that of boiling water, for instance—so that cooking cannot be depended upon invariably to render the meat harmless. . . .'

Discussing meat-poisoning, Mann says: 'It is not essential that meat should acquire a suspicious odour in order to become poisonous; certain micro-organisms render meat dangerous without producing the ordinary accompaniments of decomposition.'

In *allantiasis* or *botulism* (sausage-poisoning) a specially toxic compound is formed 'to which the term *ptomatropine* has been given on account of a certain resemblance between the symptoms it produces and those produced by atropine.'

This poison, first isolated by Zuelzer and Sonnenschein,² is attributed to a special bacillus—*Bacillus botulinus*. But in most, especially meat-putrefactions, there are many bases formed capable of producing profound toxic effect. Among such are neurine, muscarine, and choline, which Brieger isolated from putrefying flesh and fish; saprine and myoaleine, which he separated from dead bodies. All these are very poisonous, but unequally so. The most marked of the symptoms caused by the former three are, says Brunton,³ 'salivation, diarrhoea, and vomiting, dyspnoea, paralysis, preceding death; . . . even in frogs choline produces a peculiar alteration of the respiration, and dyspnoeic movements.'

'Muscarine and neurine produce in frogs a complete arrest of the cardiac pulsations, the heart stopping in diastole. . . . In mammals muscarine and neurine render the beats of the heart slow and weak, but do not usually arrest the pulsations. . . . In animals poisoned by any of these three substances the subcutaneous injection of atropine stops the salivation, arrests the diarrhoea, and removes the dyspnoea. It also prevents death from these poisons, but only within certain limits, for if the dose be very great the animals may still die. . . .' The effect of muscarine and neurine on the heart is also removed by atropine.

¹ 'Encyclopædia of Medicine' (Watson), vol. xii., p. 348.

² Cf. Brunton, 'Disorders of Digestion,' p. 282.

³ *Practitioner*, vol. xxxv., August to October, 1885.

Brunton¹ showed that the dyspnoic effect of muscarine was due to contraction of the pulmonary capillaries. The antidotal action to it of atropine, which Schmiedeberg² (who first isolated muscarine from the mushroom, *Amanita muscaria*, of which it is the active principle) had first noted, was thus explained, for atropine has the contrary effect, relaxing the pulmonary vessels.

Muscarine causes general pallor through like action on all the capillaries.

The immediate sequel of the pulmonary capillary contraction is to cause rapid engorgement of the right heart, and, if repeated, no doubt its permanent dilatation.

Whether oedema, when it follows, is due to over-relaxation of the capillaries after such toxic and abnormal contraction, I do not know. But it is at least a consequence, at times, of poisoning by sausages, fish, mussels, etc., containing ptomaines of the class discussed.

One of Bridger's alkaloids—myoaleine—causes, among other symptoms, a rise of temperature, increased pulse and respiration, excessive secretions, and finally paralysis.

All these poisons cause intense prostration before the ultimate paralysis. One, 'artificial' muscarine, got by Schmiedeberg and Harnack from choline by oxidation, and closely resembling the natural product in all other respects, has the power, like curare, of paralyzing the ends of the motor nerves.

Brunton³ compares a case of uræmia with the condition caused by a combined poisoning with atropine and muscarine :

'The secretion of urine had completely stopped; the skin, eyes, and mouth were all dry; the pupil was somewhat dilated; the pulse was beating at a rate of about 130; the mouth was held constantly open; and the breathing was laboured and gasping; but air entered abundantly into the lungs, and there was no secretion of bronchial mucus. All these are symptoms such as we find from poisoning by atropine, but in two respects the symptoms were those produced by muscarine, for the skin was pale, instead of being scarlet, as in belladonna-poisoning, and when cups were applied over the region of the kidneys, in order to restore the renal secretion, very little blood flowed from the incisions.'

The same author cites from Kaatzer a case of sausage-poisoning in a family. In all there were languor, fatigue, drowsiness, with intense dryness of the throat, and thirst. In the man there was obstinate vomiting also. On the second day the dryness of throat was worse, and the sight became affected. On the third there was paralysis of accommodation and diplopia. Next day the child of twelve years old died from oedema of the lungs. These symptoms are like those of atropine. In other cases of sausage-poisoning Brunton says, 'there are additional symptoms which point to the existence of a muscarine-like poison also. These are the presence of diarrhoea, alternating with constipation, and of colic. The pulse is also sometimes slow, small, and almost imperceptible—a condition which is typically that of muscarine-poisoning, while in atropine-poisoning the pulse is rapid from the complete paralysis of the inhibitory fibres in the vagus which the poison produces.'

¹ *British Medical Journal*, November 14, 1874.

² Schmiedeberg and Koppe, 'Das Muscarin,' p. 50.

³ *Loc. cit.*, p. 285.

In fish-poisoning, according to Mann, the symptoms 'may be purely gastro-enteric, or they may be partially or *entirely neurotic*—swelling of the tongue and face, intense itching of the skin, coldness of the hands and feet, thirst, dyspnœa, and convulsions.'

Ruxton¹ describes the symptoms of ptomaine-poisoning as follows: 'At a variable interval, an hour or two or as long as thirty-six hours, after eating tinned salmon, high game, etc., the patient complains of a feeling of nausea, restlessness, and sickness. This is speedily followed by vomiting and increasing abdominal pain, which soon becomes agonizing. Muscular cramps and clonic spasms, especially in the legs, add to the patient's misery, and soon a feeling of intense prostration, accompanied by cold sweats, nervous depression, and cardiac failure appears. The expression is anxious and even terrified, diarrhœa usually supervenes early, is profuse, watery, often fœtid, and sometimes bloody. Intense thirst is nearly always complained of, the pulse is small and quick, the urine is scanty, and the temperature varies from 101° to 103°F.'

With these pictures may be compared that of a patient stricken by pernicious beri-beri at one of those seasons when the rapidest toxic effects of that disease are seen.

Reaucar² thus vividly describes 'what happens in the acute hydropic form so common in epidemics, when one sees *phou-thoum* (Annamese = beri-beri) take on the intense type and carry off the patient, like cholera, in a few hours.

'The patient is seized suddenly with a feeling of extreme feebleness. Whatever may have been his previous state of health he is taken, as I say, abruptly, without a warning sign, with a sense of prostration, with intense dyspnœa, and constriction at the base of the chest, and, together with these, cramps in the limbs. His body swiftly swells up in a way that could only happen with œdema of vasomotor origin. . . .

'Whoever has witnessed beri-beri in that focus of its predilection, Poulo-Condore, is struck with the insistence with which the sick refer to the loss of power (*faiblesse*) to which they constantly recur, and which absorbs their attention. "Tired to the last extremity," "extreme fatigue," "ready to die," they repeat without stopping. . . .

'The victims are a prey, indeed, to real despair, which they evince by gestures as various as significant, and their fate is one

¹ 'Encyclopædia of Medicine' (Watson), vol. v., p. 412.

² 'Thèse,' 1886, 'Le Béri-béri à Poulo-Condore,' p. 57.

as to which they are so little under illusion, that now carrying their hand to their chest and now abruptly withdrawing it, as if they would snatch from thence that which was suffocating them, they ask to be delivered quickly or else to be left to die in peace. Nothing, truly, more harrowing than this picture of *foudroyant* beri-beri. . . .

'From the disease in this form none escapes; the victim is carried off in a few hours. . . .

'Of exceptional and very especial gravity is beri-beri' (Reaucar claims to have been the first to point out) 'when œdema begins in the face to the exclusion of other parts. . . . These are the most *foudroyant* cases, so much so that the death of the patient became a certainty for us when such a symptom showed itself. . . .

' . . . The pulse, at first slow and full, becomes small, irregular, undulant, fluttering. . . . Sometimes there is violent pulsation of the ventral aorta. In some the heart-beats, though hurried, are feeble. There are inconstant blowing murmurs. . . .

'This type is essentially non-febrile, but the temperature may be raised a degree or so; . . . the urine is suppressed. In some patients there is hoarseness; . . . the muscles are tender to pressure. . . . The skin is cold and wrinkled; . . . the sweat is entirely suppressed.'

For Reaucar these acute cases resemble nothing more closely than 'dry cholera.'

This note of Reaucar's was written in 1886. But cases of the type still occur (though not so freely as formerly) in our hospitals to-day.

In the writer's experience cold dry skin, dryness of mouth and throat (owing to failure of salivary secretion), are common; increased rapidity both of pulse and respiration, are usual signs early in acute cases. There is often almost complete aphonia, sometimes dilated pupils, and a widespread profound paresis of muscles, suggestive of a condition such as curare induces. Diarrhoea sometimes, oftener constipation, and frequently vomiting are complications. The latter and hiccup are of evil omen. There is intense pallor always. The urinary secretion is constantly diminished, often totally suppressed. The prognosis is based largely upon this important condition. I have seen cases with complete anuria and consequent uræmia proving fatal, which I have believed to be caused by the beri-beri toxin, but of which they have been the only sign.

The respiratory embarrassment with the concomitant cardiac distress form a condition which every observer of beri-beri from Bontius downwards has signalized as one of its earliest, most significant, and gravest features. But it would appear that most of those who have written about this symptom regard as the central feature a primary failure of the cardiac mechanism, ascribable to paralysis of its nerve-supply.

While there is in all cases some such effect, and while in some it may be the only and even a fatal symptom, this view of the crisis leaves out of reckoning two or three factors. One is, that if the nerves of the heart be first and chiefly affected, so that there is paresis of the muscle, consequent ineffectual contraction, engorgement, dilatation, and finally syncope, both sides being affected together, there should be the usual signs present which distinguish ordinary forms of cardiac weakness. But this is certainly not the case. It is exceptional in beri-beri for any symptoms ascribable to failure of the systemic circulation, such as giddiness, temporary syncope, or fainting to be noted. Secondly, the dyspnoea (the pulmonary embarrassment) precedes always the purely cardiac signs, which are first increased pulse-rate, with exaggerated and later irregular and feebler action, alteration in synchronism of the contractions of right and left sides, with reduplicated second sounds, distension of right ventricle, with final yielding of walls, insufficiency of valvular orifices, signified by corresponding bruits, great epigastric pulsation, and even venous regurgitation in the veins of the neck. These, as signs that it is the right side of the organ which chiefly, if not alone, suffers, and the establishment of the event in sequel to, but not before the respiratory difficulty, are a clear proof that it is to heightened pressure in the pulmonary capillaries primarily that the cardiac engorgement and ensuing dilatation and final failure are due.

Direct evidence of trouble in the lungs is afforded by special physical signs.

In attacks of the kind which the writer has examined these have been as follows :

The respiratory frequency is increased—perhaps to thirty or forty per minute. The second phase is shortened. Air enters freely throughout the chest. There is no cough, no pain, seldom any expectoration. The vesicular murmur is louder everywhere than usual, and harsh, tactile, and vocal fremitus perhaps a little increased. Resonance generally slightly impaired. Fine crepi-

tations are to be heard on inspiration throughout both lungs. In addition there may be extraordinary sibilations, wheezings, and sonorous rhonchi, such as characterize ordinary asthma. But there is no tendency to emphysema.¹ There is air-hunger, a sense of oppression or suffocation, pain, and great anxiety. But the face, instead of being flushed, is pale. There are at this early stage no signs of venous stasis, nor is there, even at the end, any cyanosis. Such an attack is not always fatal; it may subside as rapidly as, or even more abruptly than it appeared. Persisting, and growing more grave, frothy fluid is poured into the bronchi, and is expectorated; coarse rales attesting the œdema are to be heard all over the chest. The respirations increase in frequency, and become more laboured, the sense of suffocation grows worse, although air may be heard entering the alveoli freely throughout. To the pulmonary signs are added those of increasing cardiac embarrassment, which finally overshadow them. The patient, who remains as a rule unclouded in mind to the last, dies finally, not of asphyxia, but from syncope. The lung condition proves fatal, as it would seem, not as in congestion, because the blood becomes imperfectly ærated, but through some form of direct obstruction which it offers to the passage of the blood through its capillaries.

If it be permissible to deduce from these perhaps faultily interpreted physical signs of what may be termed the 'pulmonary crisis' of acute beri-beri the condition (hitherto not clinically recognised) of *contraction of the arterial and capillary pulmonary circulation*, we have in the event another phenomenon bringing the action of the beri-beri poison into close accord with that of muscarine.

The truth of this interpretation of the crises which characterize very acute beri-beri is supported by what is seen at post-mortem examination. The heart is then found usually empty on the left side, but engorged upon the right, and (even at an early stage)

¹ In fact, it was the prominence of this condition which led Carter (*Transactions of the Bombay Medical and Physical Society*, No. 8, 1847) at an earlier day (1845) to describe beri-beri by the title—descriptively apt, if pathologically grotesque—of 'marine asthma.' Jefferson (*British Medical Journal*, vol. i., p. 257, May 14, 1898) has vividly portrayed the perplexity of the clinician newly confronted with such a case, which presented him with the physical signs but impossible diagnosis of a total pulmonary thrombosis in a patient yet active and apparently at the moment in general good condition (the patient died within a few hours). The writer has more than once seen a similar condition taken for the first stage of pneumonia.

softened and dilated. The lungs, pale and anæmic, generally show some œdema, and if the agony has been prolonged some hypostatic but never any general congestion. There may be also (as Wright has noted) acute emphysema. The spleen is invariably softened and enlarged; the liver may be so also; there are petechiæ on the pleura, on endo- and peri-cardium, hæmorrhagic spots, and extravasations under the mucosa of stomach and duodenum, sometimes throughout the intestine, and (Mott¹ says) similar but small foci in the liver. H. Wright observes also glomerular nephritis. These findings are such as are found also in the various ptomaine-poisonings.

In the symptom-complex of beri-beri, then, it may be said, there is just such a picture as might be expected to be presented as the result of intoxication by a ptomaine or alkaloid. While it must be left to the future to isolate and to determine exactly the properties of the poison, it may now at least be inferred that it presents close resemblances to known alkaloids. Like *morphia*, it has an early stimulant, and, later, depressant effect on motor nerve-ends and motor spinal cells—early hypermyotonus, exaggerated tendon reflexes, and later loss of these; it produces brief stimulation, and later depression of sensory nerve-ends and brain (occasional early hyperæsthesia, later languor, prostration, anæsthesia). It resembles *atropine*, in depression of both motor and sensory nerve-ends (paresis, anæsthesia), in depression of secretions (dry mouth and throat, suppressed sweat and urine), removal of vagus inhibition (tachycardia), early stimulation, later depression of vasomotor nerves (pallor, followed by œdema). There is likeness to *cocaine* in the alteration of sensory nerves (paræsthesia), depression of motor nerves (paresis), and diminution of muscular output (feebleness).

In several of the respects mentioned there is great resemblance to the action of the animal alkaloid, *ptomatropine*; while, in the capillary-contraction effect, that of *natural muscarine*, and in the intense prostration and paresis (paralysis of motor end-plates), the actions of *artificial muscarine* and *curare* are imitated.

Seventh Conclusion.—*The beri-beri poison is probably an alkaloid which is stable and non-volatile, and resembles atropine and muscarine in some of its effects.*

8. Mode of Production of the Poison.—The next most interesting question is, *How* is this deadly poison formed in what was previously wholesome material?

¹ *British Medical Journal*, October 28, 1905, p. 1099.

All the organic poisons, as most other compounds found in Nature, arise as the result of the action of living cells, being sometimes formed and retained within them, sometimes so formed and secreted—diffused—into surrounding media, sometimes formed only in the media through the action of other bodies first secreted by the cells. The processes by which the poisons are produced are, in their intimate or ultimate nature, being vital functions, not of course understood. But so far as their phenomena can be arranged they appear to depend, whether the products are without or wholly within the cell, on the mediate operation of what are called 'ferments,' or 'enzymes.' All the phenomena of fermentation, of 'decomposition,' or 'putrefaction'—which is only complex fermentation—depend on these; and it may, therefore, be of use, in a speculation as to the mode of formation of the beri-beri poison, to refer to the properties which distinguish them.

'Ferments, or enzymes,' says Hahn,¹ 'are substances of a proteid nature which have the power of inducing chemical change in other substances, whereby there arise bodies of lesser caloric. Such a change occurs without the enzyme itself forming any lasting chemical combination, either with the substance which is being broken up, or with the products of disintegration. Thus in general there is no using up of the enzyme.'

'The enzymes are active only when in solution, but not when in a dry condition; they are not destroyed by drying, but they require a sufficient quantity of water for their action. The activity of solutions containing enzymes is, without exception, destroyed at the temperature of boiling water.' But their action is arrested by temperatures much lower. Most ferments are rendered inert when in solution, or moist at 70° C. Cold does not destroy them. For each there is an optimum temperature, at which the activity is greatest. When quite dry, again, they can be heated beyond boiling-point—120° to 160° C.—without losing their active properties, which reappear when they are redissolved. Some can act only in an acid, others only in an alkaline medium.

The action of the enzyme is commonly one of hydration; organic, highly complex bodies are split up into simpler ones by the introduction of water into the molecule. Thus, starch and allied bodies are converted into fermentable sugar. Enzymes of this class are termed 'diastatic.'

¹ 'Encyclopædia of Medicine' (Watson), vol. iii., p. 272.

The ferment of barley malt is the best known of these. But a host of such ferments exists in Nature, and they are found in parts of most growing plants, especially in seeds at germination. They are formed and excreted by many of the lower fungi (*Aspergillus niger*, *A. coryza*, *Penicillium glaucum*) in yeasts (*Torula*), and many bacteria. In the secretions of animals (saliva, pancreas, liver, blood, etc.) are to be found enzymes of similar action.

Other ferments split up cane-sugar and other polysaccharides—inverting ferments. The compounds called 'glucosides,' which form the specific secretions of many plants, can be fermented by enzymes, which are often found in association with them. Thus, amygdalin, the native glucoside of sweet and bitter almonds, is turned by the enzyme *emulsin* into sugar, hydrocyanic acid, and oil of bitter almonds. *Myrosin*, another enzyme of this class, is found in watery extracts of white and black mustard-seeds. It breaks up the myronate of potassium contained in black mustard-seeds into mustard-oil, sugar, potassium sulphate, and some free sulphur.

Other enzymes dissolve albumin—proteolytic enzymes. In general most animal and vegetable cells contain such. In most cases the enzyme is produced, remains, and acts only on albumin brought to it within the cell (endoferments); but in other cases the enzyme is excreted. Various processes of physiological digestion are carried out by such means. The products, peptones, albumoses, amido-acids, hexon bases, leucin, tyrosin, etc., are, in a sense, toxic. Fermi has shown that all those bacteria which liquefy gelatine—e.g., the cholera vibrio, the anthrax bacillus, the pyogenic staphylococci—liberate peptonizing ferments during growth.

'Urinary' enzymes convert urea into ammonium carbonate. The *Micrococcus ureæ* was formerly supposed alone able to do this. But Miquel has recently shown that sixty different bacteria, cocci, bacilli, sarcinæ, cultivated from the air, river-water, and drainage possess this power, their action being due to an enzyme—*urase*—which may be separated from them.

The 'monosaccharide-splitting' enzyme, converting glucose and other sugars into alcohol and carbonic acid, to which the action of the yeasts in beer, wine, and vinegar manufacture is due—*zymase*—may in like manner be obtained separately from the cells (E. Buchner and M. Hahn). The *zymase* extracted from yeast cells (crushed and ground up with quartz-sand and subjected to hydraulic pressure) in a dry condition will stand heating to 100° C. *Torulæ* which have been carefully dried and then heated to 120° C. so as to kill them are still capable of producing fermentation, a proof that their *zymase* is a content of the cell, an endoferment. Oxidizing and reducing ferments are supposed to occur in the blood and tissues of animals, fresh extracts from which convert, for instance, benzyl-alcohol into benzoic acid, and decompose peroxide of hydrogen.

'There is no doubt,' Hahn says, 'that certain of the bacterial poisons—tetanus poison, for instance, which in extremely small quantities acts with great power on the animal body—are closely related to the ferments.'

The proteolytic enzymes of bacteria are apparent in cultures by the liquefaction of gelatine. They are distinguished from

most other enzymes found in the vegetable kingdom by being efficacious only in alkaline solution. They are rapidly destroyed by weak acids, likewise by heating in water to 70° C. Sunlight reduces the power of this ferment. Its elaboration continues only so long as the vegetative power of the bacteria persists. But the excretion of it may continue after the death of the bacteria from the old, dead, or degenerated cells of which it freely escapes.

It is through the direct action of the enzymes they secrete that the bacteria bring about decomposition of the media in which they occur. The new chemical compounds thus brought into being, together with others—the products of the interior activity of the organisms, as cells, which, excreted, diffuse into the surrounding medium—make up the 'products of decomposition.'

Between fermentation and putrefaction, says Nuttall,¹ 'there is no essential difference, . . . the difference being but one of degree.'

The various forms of decay vary merely in the number and complexity of the organisms at work, and their products.

As a result of physiological activity certain bacteria form products which may be extraordinarily poisonous. These may remain within, or (as in the case of the tetanus and diphtheria bacilli) diffuse into the surrounding medium from the cell. They are termed *toxins*.

The toxins are, says Nuttall,² 'unstable compounds which are injured by heat, light, and various chemical agents. Their chemical constitution is highly complex, and remains to be determined. It has been proved that they are not ptomaines, basic substances, nor proteins.' Of tetanus toxin, it is estimated that $\frac{1}{2500}$ grain would be a fatal dose for a man of average weight.

Since the production of poisons in all these cases depends ultimately on some form of fermentation, it may seem at the present barren to inquire whether that on which the beri-beri poison depends be a ferment native to rice-seeds or a ferment secreted by some saprophyte of the grain, or whether the poison is, like the bacterial toxin and the vegetable alkaloid, an intracellular product of the posited saprophyte.

But the question has this important bearing in epidemiology, that if it be a ferment only, the original noxa cannot be spread, if capable of spread at all, with any facility from infected to non-

¹ 'Encyclopædia of Medicine' (Watson), vol. viii., p. 82. ² *Ibid.*, p. 84.

infected material by any means other than direct transfer of rice containing it. Moreover, a necessity of all fermentation being the presence of water, it would follow, were the noxa simply of this nature, that its operation would be impossible in material kept dry.

On the other hand, let the process be the result of any form of bacterium, or of fungus able to propagate by spores, and it becomes evident that, provided a suitable medium offer—and all cereals are almost identical in the nature of the materials which compose them—the production of beri-beri poison may be set up in numerous substances as a result of infection by any of the accidental modes upon which infections commonly depend.

There are many things which render the conception a plausible one that the beri-beri poison is the result of a ferment native to, and only found in, rice-grain. One of these is the fact that the poison is only found in rice. Were the poison due to an epiphytic or parasitic sporing fungus, or a bacterium, it would seem almost impossible that the same agent should not just as often, or if not as often, at least sometimes, attack the other cereals, the composition of which is so like rice.

It must be admitted that epidemics of beri-beri ascribable to any source other than rice are of the rarest happening, if they ever do occur. But it will be seen that a spore-bearing fungus or a bacterium is not the only alternative to a native rice-ferment.

The facts, again, that boiling kills the precursor of beri-beri poison in rice, and that, as general evidence shows, the poison appears to be most extensively produced when rice is allowed to get wet, which would favour the ferment view, no less support the theory of any other fungus being the agent, for all bacteria and fungi are favoured by moisture.

On the other hand, it would seem to be certain that, however much the production of poison in stale rice may be favoured by damp, it also occurs in samples which are, and always have been, kept quite dry.

When the fact is recalled that ferments act only *in solution*—*i.e.*, under conditions implying much more moisture than ordinary circumstances of damp can supply—the probability of the toxic agent in rice being a simple ferment becomes very remote.

Another consideration supporting the rejection of ferment is, that in all the fermentative processes which have been studied, the products are almost always not single but many. Various new bodies arise in the course of all fermentations, which are

revealed in alterations of texture and consistence, of colour, of taste, and odour, in the materials acted upon. But many samples of rice which have been productive of beri-beri, and have therefore contained its poison, have been, according to reliable evidence, quite free of all changes which could suggest alteration of their chemical composition, such as those described, which in some degree or other it would seem that every fermentation must produce.

The toxic change in stale rice, therefore, cannot with any probability be ascribed to any *simple ferment*.

Similar reasoning leads to the rejection of a bacterially-derived enzyme.

While the condition as to *solution* does not apply to bacteria, it is certainly the case, with the putrefactive forms at least, that their free growth demands a free supply of fluid, while the activity of all species is dependent upon water, ceasing in the absence of moisture, and being destroyed by complete desiccation.

The products of bacterial growth, too, are always complex, various compounds being liberated due to the enzymes they secrete.

Finally, in spite of much research, no evidence of changed chemical composition in stale rice has been submitted, no attribution to such rice of any special bacterium has been made.

The production of the poison through any fermentation of the material, the presence of any special bacterium as its source, having alike been rejected, there remains only the possibility that the toxic agent is some obscurer fungoid organism, itself poisonous, specifically associated with the grain. Such an agent, while perhaps growing in and at the expense of the seeds, need entail no general alteration in their physical consistence or chemical composition, the poison being not an extra-, but purely endo-cellular product, the result of the parasite's own specific or physiological activity, and the alteration of medium in which the body occurs being limited to that part of it actually consumed.

Eighth Conclusion.—*The formation of poison in stale rice is probably due neither to fermentation nor to bacteria, but to the growth in it of a special fungus.*

9. **Distribution of Fungus in the Seeds.**—The position at which we have now arrived is that the poison in stale rice is some specific fungus itself toxic. It was shown earlier that fresh rice contains no poison, or too little to produce symptoms. It was also

shown that the poison in stale rice is yet derived from the fresh seeds, since simply boiling or heating the latter prevents the formation of poison in them subsequently. Hence it may be inferred that the difference between the fresh and stale grains is quantitative merely, not qualitative. The toxicity of the latter is but the result of extension of a growth already present.

But the facts lead to the further inference that the fungus so derived from fresh rice is perhaps not a parasite, but merely an epiphyte of the grain ; that it resides, at all events, rather upon than within the fresh seeds, never largely invading their texture, —at least, until decorticated.

The reason which appears to make this plain is that, as has been stated, collections of rice-seeds, when fresh (or preserved, by curing, in the same condition, as regards possible specific toxicity, as they were in when fresh), are never poisonous.

Were it the case that the fungus present in fresh rice-seeds or raw padi could invade and displace the substance of the seed (as ergot), there would be, so far as known analogies go, no limit to the extent to which the seed should be so destroyed. The whole of it might be, and probably usually would be, sometimes thus rendered poisonous. But what applies to one seed may do so to more. Thus, the greater part or even the whole of a given crop of grain, might become spoiled by the fungus.

But this never occurs, since, as so often insisted upon, neither fresh nor cured rice ever show evidence of such toxicity. What applies to collections of seeds apply equally to the component seeds ; it must be assumed that neither are these, individually, ever invaded by the fungus. The latter, therefore, though of the seeds is not in the seeds. The fungus is present in fresh rice, it must be inferred, only to a small extent, and is present not in the substance of the seeds, but only among them, or upon their surfaces.

The parasite, endophyte, or epiphyte, may grow anywhere within or on the envelopes of the padi-fruit ; it may be even extensively spread amongst these, yet leave the seed, the endosperm, not at all affected.

In this manner might be explained the paralysis of padi-fed horses (equine beri-beri), as a natural result of consuming, as the horse does, with the freshly crushed grain, its envelopes also.

The reasoning followed so far, therefore, indicates that the ultimate 'berigenic' factor is some sort of fungus closely associated with raw rice-seeds, not during life penetrating their

interior, but growing freely within the grains as a saprophyte when they have become devitalized on decortication.

Ninth Conclusion.—The beri-beri producing fungus of rice is probably a surface-parasite or epiphyte affecting the seed saprophytically after decortication.

10. **Similar Fungi found in other Grains—Rye and Darnel.**—For the fungus which has been postulated as occurring in padi and rice, and causing beri-beri, at least two described fungi provide analogy, found upon other grains, which they also render toxic.

One of these is peculiar to rye, the other is the seed-fungus of the darnel.

Sickness as a result of consuming rye vitiated by a fungus other than ergot had been described by Eriksen¹ in Sweden in 1883, and later, in 1891, in South Ussuria (in Russia) by Woronin,² and in France by Prillieux and Delacroix,³ who identified the fungus, and named it *Stromatinia temulenta*.

Prillieux⁴ describes the toxic symptoms as resembling those caused by darnel, except that they are more intense and rapid. The condition produced seems to have been one of giddiness, great restlessness, finally prostration and torpor. Similar effects were produced in dogs, pigs, and fowls.

'The poisonous grains are of very poor appearance—small, light, and wrinkled, like grains which dry up before they are full grown.'

'On transverse section, it is seen that the outer portion of the albumin of these grains is invaded by the mycelium of a fungus, which forms a thick matted layer, taking the place of the normal tissue, which is destroyed. No trace is to be found, as a rule, of the outer layer, which in the normal seed is composed of cubical cells with a content exclusively of proteid grains.' The starch-cell layers nearer the surface are also corroded, and here and there filaments of mycelium project into the envelopes of the grains. If affected seeds be kept moist at 15° for about a fortnight, white outgrowths formed from these filaments in a sporing state appear on the surface. The spores are formed and extruded three or four in succession from the interior of branched conidia springing direct from the mycelium. This sort of spore appears at a time when the mycelium of the fungus is yet con-

¹ Reference : Guérin, *Journal de Botanique*, 1898, No. 15, p. 237.

² *Ann. Agronom.*, 1891, t. xvii.; and *Bot.*, ii., 1891, No. 6.

³ Prillieux and Delacroix, *Bull. Soc. Bot.*, 1891., t. xxxviii.

⁴ 'Mal. des Plantes Agricoles,' vol. ii., p. 454.

fixed to the surface of the seed. But if such seeds be left in the moist chamber for several months, the mycelium, having by degrees penetrated throughout and replaced all the seed, a second form of fructification appears in filiform paraphyses, and large apothecia springing from the mycelium. These forms appear from October to December. Grains of rye giving rise to these apothecia are larger than ordinary, owing to the swollen mass of filamentous fungus which they contain, nothing of their original content remaining except a few small and partly corroded grains of starch.

Supposing a grain of rice to have been replaced by a parasite of this description, which one may further suppose, at the same time, to be white or colourless, so as to be indistinguishable by the eye, odourless, and tasteless, yet toxic, then we should have the state of affairs postulated as happening in regard to the rice which causes beri-beri.

The poisonousness of the darnel, says Guérin¹ (*Lolium temulentum* and *vars.*), has been known from very remote times. Guérin says Plautus, Vergil, Ovid, Livy, all refer to this property. Cattle have become sick and sometimes died from eating it. Men have suffered from its effects when accidentally mixed in flour or fermented liquors.

The symptoms are giddiness, vomiting, prostration, paralysis of ocular muscles, and coma. A horse has been killed by 2 kilos of the seeds.

Antze, in 1890, isolated from darnel two alkaloids—*lohiine* and *temuline*—one of which he stated to act as a nerve-poison, the other as a digestive irritant. Hofmeister, in 1892, found but one alkaloid—*temuline*—a crystallizable body, exercising specific toxic effect on the nervous system. The irritant action on the alimentary canal he attributed to certain acrid oily matters also contained in the seeds.

Guérin describes his own discovery of the peculiar parasite of darnel as in a sense accidental, the fungus having been encountered in 1898² in the course of a systematic research upon the seed-envelopes of grasses generally. 'Our attention was attracted to a sort of felt-work lying between the aleurone layer of the seed and the hyaline layer, representing the remains of the nucellus. This tissue occupies the whole surface of the grain. By direct inspection of the layer, when the various envelopes

¹ *Journal de Botanique*, August, 1898, Nos. 15, 16, p. 230.

² It was also independently described by Vogl in the same year.

of the seed are separated, it is easy to perceive that one has to deal with filaments of mycelium. The hyphæ of this fungus-stratum are colourless, closely interwoven, and more or less twisted on themselves.'

Investigation of a very large number of samples of darnel from all parts of the world, and of different varieties, showed that very few plants were free from the parasite. 'As a result, of over forty specimens from the most varied sources, only three appeared to be devoid of the mycelium.' These came from places from two of which other samples taken were, however, affected. 'It is to be observed that, in regard to any given locality, if one grain is *parasite*, all are.'

Guérin concluded that in *L. temulentum*, *L. arvense*, and *L. linicola*, all reputed poisonous sorts, the parasite was practically constant. In *L. italicum*, not reputed as poisonous, it was not present. He compares the fungus with that of toxic rye, and decides it to be of a different species. Among obvious differences between affected seeds of the two plants noted by Guérin were the alteration in the aspect and size of diseased rye-seeds, the parasite of which replaces largely their proper substance. The lolium seeds, on the contrary, showed no deformity; they germinated excellently, the fungus in them never being found near the embryo. Whereas in rye-seeds the fungus destroys all the aleurone layer, in lolium, Guérin says, nothing of this kind happens. The diastatic (gluten) layer always has all its cells intact, and the fungus never penetrates the endosperm.'

The general correctness of Guérin's account is confirmed, with modifications of detail, by Freeman, from a more recent article by whom I extract what follows:

'In 1898,' says Freeman,¹ 'attention was drawn to the fact that a large percentage of the grains of this grass contained a definite fungus mycelium, always situated in a definite layer of the seed—*i.e.*, in the remains of the nucellus just outside the aleurone layer of the endosperm.'

'It has long been known that the grains of the darnel contained a poisonous body (lolium) which can be extracted by ether. This substance has marked toxic action on rabbits and certain carnivorous animals, and is said to induce vomiting and other unpleasant symptoms in man, but to affect hogs, cattle, and geese but little or not at all.'

'All attempts to grow the fungus outside its host plant or to

¹ *Philos. Trans.*, Bd. 196, 1903, p. 1.

induce it to form spores have failed. . . . The evidence so far points to the present case being an interesting example of symbiosis. . . .

‘The cross-section of the average grain of *lolium* shows a layer of densely-woven fungus hyphæ just outside the aleurone layer on the crushed remnants of the nucellus, of which the outer cell-walls form the “hyaline layer.”

‘The proportion between the grains with, and those without, the fungus is exceedingly variable.

‘In one package of grass grown at Cambridge 15 per cent. were devoid of fungus, and in a package received from Upsala, out of ten grains examined, eight did not contain hyphæ.

‘The hyphæ may, under favourable conditions, penetrate through the aleurone layer into the starch endosperm at any point, and even penetrate to the centre of the seed.

‘When the embryo of the grain resumes growth on germination, the hyphæ already in its tissues . . . keep pace with the growth, and can be detected in the growing point throughout the life of the plant. . . .

‘Some weeks before the spicate inflorescence emerges from the enveloping leaf-sheaths its initial inception exists as a short cylindrical mass of merismatic tissue, embossed with the rudiments of the floral parts, glumes, and paleæ. . . . Sections show that almost the entire mass is permeated by the fungus hyphæ, so that it seems very improbable that a single ovule need escape infection. . . . The ovule is infected by way of the funicle, and the hyphæ extend into the bases of all glumes, paleæ, and plaments . . . The rudiments of the ovules contain hyphæ from the earliest stages. . . .’

In both these parasites of the rye and the darnel we have, then, examples of fungi which are toxic, which are specifically associated with particular sorts of grain, and which are practically in each case confined, so long as the grain is raw or fresh, to the surface of the seeds

Were the grains either of rye or *lolium* prepared, as are those of rice, for food, by complete decortication of their envelopes, and scouring off the proteid layer, it is obvious that the fungus affecting them would be almost entirely removed. The unhulled grains, still retaining the poison among the envelopes, might be poisonous to animals consuming them, as padi is to horses; while the fresh seeds, newly stripped and deprived of the fungus, would be innocuous, as is fresh rice. But ordinary processes of

hulling, however carefully conducted, could not divest the grains of every particle of the fungus once investing them. Some of it would, minutely divided and generally distributed, remain among the mass of grains. It has only to be conceived that such particles should be able to invade by growth the seeds with which they are in contact—as there is evidence the fungus of rye actually does—and it would follow that the stripped seeds would become in course of time, and under suitable conditions, as toxic after as they were before decortication. Were the toxic but unhulled seeds (darnel or rye) heated before hulling sufficiently to destroy their fungus, then the seeds, freed from the parasite, and so made non-toxic upon decortication, would (unless infected from some fresh source) remain so. There would thus be produced the phenomenon of a grain (rye or lolium), poisonous when raw and with its envelopes, but not so when hulled and fresh; poisonous when uncured and stale, but not so when cured, however stale; the result of a specific fungus growing upon the surface of the seeds.

In some such mode, and through the action of such parasites as those affecting lolium and rye, all the analogies seem to indicate, then, rice is rendered poisonous so as to induce beri-beri.

Though not yet recognised, perhaps not visible to naked eye or even to microscope, the toxic agent lurks within the envelopes of the seeds. Dislodged thence in milling, and freely distributed by that process among all the grains, it attacks them, growing freely as a saprophyte in the dead material. The rice thus infected becomes increasingly toxic as it grows more stale. All those conditions—of darkness, of warmth, of moisture, of disintegration—which promote the growth of other fungi favour also this, which is but a special form of decay. But if taken fresh, before such change has advanced, the rice is harmless. If the grain is boiled before it is stripped, the fungus in the grains is killed, and, whatever other form of decomposition it may undergo, the specific poison of beri-beri is, in such cured rice, never formed.

Tenth Conclusion.—*The specific fungus of beri-beric rice is, like that of toxic rye and lolium, probably a parasite affecting the surface of the seed.*

Some Further Deductions from Seed-Fungus Theory.—Accepting the view that the agent which produces beri-beri is a fungus such as has been suggested—a surface-epiphyte or parasite of rice-seeds, which becomes saprophytic on them under conditions promoting decay—deductions follow which serve to

explain certain difficulties and some important features in the natural history of the disorder.

One of these deductions is that, whatever the quantity, absolute or proportional, of toxic fungus in the raw grain or unhusked padi, the amount in grains which have become saprophytically invaded later must be far more. A given weight of stale rice will thus contain, ordinarily, more poison than the same weight of raw padi.

(a) **Equine and Avian Beri-beri.**—In this fact may lie the explanation of the discrepancy which there appears to be between the liability of horses and fowls respectively to beri-beri.

In the text it has been mentioned that horses fed on raw padi acquire a form of paralysis clinically indistinguishable from human beri-beri; while the statements of Eijkman, Sakaki, Rest, Hose, and others, proves that a like disease attacks fowls fed on stale, or, exceptionally, fresh, decorticated rice, but not fowls fed on padi. A legitimate inference from this last fact would appear to be that padi, like fresh rice, contains, as a rule, no poison. But this would directly oppose the view that it is a poison in padi which causes paralysis in the horse.

The seeming contradiction is, however, simply explained if we suppose that the sensibility of the fowl to the poison is less than that of the horse, as it undoubtedly is for some other poisons—*e.g.*, belladonna. It would follow that, as is the case, horses would succumb to the small proportion of the poison found in padi, though fowls did not, and that fowls which resisted the dose contained in a given quantity of padi would yet be poisoned by a smaller quantity of the more toxic cleaned rice.

Moreover, it may be observed that the demonstrated facts as to fowls are merely that they derive paralysis from stale and (exceptionally from fresh) white rice, but not from *padi*, in the quantity ordinarily given as a ration for subsistence. It is therefore possible that *were a sufficient quantity of padi given*, so that the actual weight of poison eaten (proportionately much less in padi than stale rice) should be equal to that consumed in a diet of stale rice, the same result would follow—fowls would be found to get paralysis from padi just as horses do.

On the other hand, horses are never fed on cleaned grain. It is impossible, therefore, to deny force to the speculation, otherwise highly probable, that they *would* derive the disease even sooner from cleaned stale rice than from padi—*i.e.*, be poisoned by a less weight of it than of padi.

(b) **Discrepancies in Epidemiology to be Expected from Surface-Epiphyte Theory.**—The peculiar distribution of the poison postulated in grain, while explaining all the conflicting facts observed as to the distribution of the malady, suggests the occasional occurrence of other discrepancies, of which, when attention has been drawn to this subject, examples may be brought forward. It should thus be likely that, in course of time, under damage, moisture, etc., the external epiphyte should invade the seed even *before husking*, so that rice derived from such padi would be toxic even though eaten fresh. This was the case in one of Sakaki's experiments (*h*), in which *fresh* rice made from seven-year-old padi proved toxic.

Many of the instances recorded by Fiebig of beri-beri occurring among Malays of the archipelago, whose diet was customarily one of fresh rice, own, it is possible, a similar explanation, although in their case the more obvious ones have also to be reckoned with, of a lapse from use of fresh rice, or an unwonted increase in the proportion of it taken.

(c) **Biology of Rice-Fungus a Factor in Periodicity.**—Prillieux observed that the second (apothecial) form of fructification of the ryé-fungus appeared only several months after reaping the corn, and at a particular season—the autumn. Such a characteristic, did it apply to the padi or rice-fungus, would tend towards establishing a corresponding periodicity in its effects in epidemics of beri-beri.

Thus, another is added to the numerous factors which already complicate the epidemiology of the disease, but at the same time serve to render intelligible fluctuations in its prevalence which would otherwise go unexplained.

(d) **Essential Nature of Toxification of Stale Rice an 'Infection' of Sound by Diseased Grains.**—It follows from the conception of the 'berigenic' noxa as an agent which, derived from one part, or one grain of rice, gradually invades more, that the process is, in a sense, an *infection*. Granting that from one portion of rice, the *addition* making it toxic may spread through a larger mass, it becomes a mere matter of time for a single grain, a particle of diseased rice, however small, or even a solitary spore of the postulated epiphyte, to render entirely toxic any mass of rice, however large, into contact with which it may be brought under otherwise suitable conditions. The minutest quantities of the epiphyte (in this respect resembling any other contagion) derived from old diseased samples of rice, and left in bins,

bags, and store places, may thus gradually spoil originally good rice.

In this sense, and in this way, a certain sort of 'place-effect,' or infection would be established. But the infection would not act directly upon the patient, nor indirectly, through the operation of an '*emanation*' which it should produce, but merely through alteration of his rice, much as milk, for a familiar example, *kept in particular places*—vessels or dairies—is more wont to turn sour in them than in others.

(e) **Possibility of Beri-berigenic Change in other Aliments than Rice.**—Another consideration is this: the agent, being able to penetrate and render toxic the hard mass of cells stuffed with starch-granules which constitute a grain of rice, the invasion of other and softer cereal food-stuffs by it must be yet more easy. Thus it becomes again in the highest degree probable that *alimentary substances of all kinds* made from flour, or similar to rice in their chemical composition, *should be able to undergo the same beri-beric change* as rice does, when casually infected by the same agent.

This would, of course, be especially likely to happen in rice-eating countries, and when the presence of the disease shows its cause to be abroad.

(f) **Spread of Berigenic Agent in New Countries.**—But granting to the infecting agent a moderate amount of vitality, it might conceivably be carried to places where rice itself was not, and result in the production of beri-beri through channels which would not be traced because they would never be suspected.

(g) **Poison probably Variable.**—What the exact *composition* of the *poison* may be, it must be left to future labours of chemist and mycologist to determine.

The two different types of disease—the 'dry' and the 'drop-sical'—the greater prevalence of the cardiac symptoms (*shiyo-shin*) in some epidemics, or of the skin troubles ('*pirri-pirri*'—*cf. kriebelkrankheit*) in others, suggest that the composition of the poison, like that of ergot, is compound, and variable. A very common feature in all epidemics—namely, that near their termination more persons are attacked, but in slighter degree—seem to argue that the poison as it becomes older also diminishes in vigour—a characteristic which ergot shares.

(h) **Most Prone to attack Rice, but not Peculiar to Rice Alone.**—As already pointed out, in formulating the theory, if such an agent can invade rice, the analogy of many other vegetable

parasites renders it *improbable that it should be confined exclusively to that grain*. It will be likely, under favouring circumstances, to attack other cereals, which may thus become a cause of beri-beri equally with rice. Rice is the common, but not the *only*, vehicle of the disease. As already set out at length, beri-beri is to be conceived of more generally as a *grain-poisoning*.

Ergot of rye is by no means confined only to that grain. 'It appears,' Van Boeck states, 'not on rye only; it is found in rainy summers in great abundance on wheat, barley, *rice*, millet, brome-grass.' It may attack, in fact, any of the gramineæ. Accordingly, epidemics of ergotism have been recorded in other than rye-eating countries. The last great epidemic in England was in 1109. But cases of gangrenous ergotism were described here, Aitken says, by Wollaston (*Philos. Trans.*) in 1762. The same authority states that Sir W. Wilde, of Dublin, described cases as occasionally happening in his time in Ireland.

The 'berigenic' agent residing in rice, then, whether single or manifold, if it resemble ergot, though commonest upon, is probably not confined to, that grain. As there is no *a priori* reason why it should not, so the ergot analogy makes it likely that it should invade at times other cereals, the consumption of which, when affected, would explain epidemics in which the action neither of plain rice nor its derivatives could be traced.

The views thus elaborated as to the nature of the beri-beri agent, and the probability of its infecting other articles of food and other cereals, so simply and sufficiently explain all the known facts as to the production of beri-beri that they may be accepted as representing, if not the whole, at all events very nearly the whole truth.

It remains to show how the various objections which have been from time to time levelled against *rice* as the possible cause of beri-beri may be disposed of.

SECTION VIII

OBJECTIONS TO PREVIOUS RICE THEORIES—BERI-BERI IN TEMPERATE LATITUDES—NOTE ON 'ARSENIC AND BERI-BERI'

ALL the objections hitherto recorded against *rice* as the source of beri-beri were made before much of the evidence which has been set out in this work was available, and were addressed to quite other theories as to its mode of action than that which has now been formulated. Few of them, indeed, could have been raised had some of the facts now presented been earlier known, with the inferences which they justify. For nothing in the epidemiology of beri-beri, so far as the writer is acquainted with it, conflicts with the theory as now defined.

Most of the exceptions taken have been based on instances where either (1) rice which should have caused beri-beri, according to the observer's judgment, has failed to do so ; or (2) where rice which ought to have been innocuous has produced the disease ; or (3) where beri-beri¹ has appeared although no rice at all was eaten.

As before, it may conduce to clearness to summarize the factors or conditions on which beri-beri from rice demonstrably depends. These are :

1. Sort of rice, whether (*a*) Cured, or (*b*) uncured (fresh, or red ; stale, or white).
2. Quantity of rice eaten (absolute, relative).
3. Time during which rice is consumed (not less than six months usually necessary).
4. Personal idiosyncrasy (whatever the poison, some will resist it better than others).

It is clear that rice, as a cause of beri-beri, in particular cases,

¹ *I.e.*, real, or merely asserted.

cannot be excluded unless, all the circumstances being investigated, it can be shown that there were present all the conditions indicated above as necessary for it to produce effect—*i.e.*, it must have been of such sort as could be toxic, have been taken in sufficient quantity, and for a sufficient period. Even then, failure to get the disease will prove nothing in an individual or a small community, since in most epidemics, although many may be attacked, yet many, too, escape. The upper grade prisoners in the gaols who, although exposed longer than any others to the cause, yet remain free, are instances in point.

In cases where beri-beri occurs, but the direct use of rice cannot be traced, it must be further shown, before it or its influence can be proved to be excluded :

1. That rice is not used in another form—as, for instance, ‘arrowroot,’ or as an adulterant of flour, or as glucose, starch-sugar, which often is practically an extract of rice.

2. That the food in use has not been exposed to contact with, and possible contamination by stale toxic rice, as might happen with flour kept in old or dirty bins.

Where these sources can be negatived, we have excluded the agency of rice, indeed, but not that of the rice-poisoning agent which, usually associated with it, may have, in the particular instance in view, gained access to other grains. Beri-beri is still due to the same noxa ; is still a grain-poisoning.

Finally—and perhaps this should have been adverted to first—not every epidemic of polyneuritis closely resembling (as all epidemics of the nature must do) beri-beri—which is but one form of it—is to be too readily accepted as the real disorder. There are, as has been earlier noted, several maladies simulating the tropical malady for which they have been only too often mistaken. Of these, more will be said later on.

The commonest sort of objection raised against rice has been that, among persons or communities sharing exactly the same rice, perhaps out of the same pot, the distribution of the disease is unequal. Some members of a party, some among the communities, get it, while others escape.

The attendant circumstances, as the period of consumption, and especially the relative proportion in which such rice is consumed by each class, the accessories eaten, slight alterations in which have been shown to be so important in preventing the disease, are usually in these statements ignored.

1. **Some Objections by Travers.**—Thus, Travers¹ has urged the case of three institutions in Selangor—a lepers' asylum, a home for incurables, and the criminal prison—at each of which the inmates received daily the same sort of rice, distributed from the same store, and supplied by the same contractor. At the criminal prison beri-beri was constant and severe, among the inmates of the other places no cases occurred. If rice caused the disease at the prison, why not, asks Travers, among the lepers and incurables also?

At all these institutions many of the individuals, he states, were fed on the same rice for an equal and sufficient length of time—over six months.

The explanation here clearly lies in the *relative* quantity eaten, which is less in the hospitals than in the prison dietary. In the diet of the lepers, as in that of the patients of all the hospitals, there is more variety, and fresh meat, eggs, or fish are given every day, which is not so in the prison.

2. Ellis² gives the following remarkable account: 'An instructive instance came under my notice in 1900, entirely, I think, putting Siam rice out of the question as a cause of beri-beri. A well-found steamer containing 28 Malays and 51 Chinese sailed from Singapore for New Zealand and Australia. All were fed on Siam rice, cooked in the same manner, and in one galley. When in cold weather, near New Zealand, beri-beri broke out in the starboard fore-castle, inhabited by 14 of the Malays, and in all there were 8 cases and 5 deaths. This fore-castle had the galley situated immediately aft, with but a thin wooden partition. The heat from the galley caused the cabin to be always sweating and steaming, as it was somewhat wet from the bad weather experienced at the time, and, in fact, converted into a perfect incubation chamber. . . . No food was taken to or consumed in the fore-castle.'

Ellis, being consulted, recommended disinfection, and the abandonment of the fore-castle. He does not say if this was actually carried out.

'No further cases occurred after these. . . . Had the rice been in fault,' he says, 'it is difficult to understand why the eight cases occurring should have all been among fourteen men occupying one cabin, and that the remaining hands should have entirely escaped.'

¹ *Journal of Tropical Medicine*, September 15, 1904, and local publications, for which see Bibliography.

British Medical Journal, November 14, 1903.

But, the cause being ascribed to infection derived from the one 'incubation chamber,' it is at least as difficult to understand why, of the 14 who occupied it, 6 entirely escaped. Why, out of this cabin, or out of the whole crew of 51 Chinese and 28 Malays, only these *eight* particular men were attacked the facts given are not sufficient to enable us to determine. It may have been that they had been longer on such a diet, having shipped for this just after coming off another cruise, or that they ate absolutely more rice,¹ or relatively more, having less of some other accessories. This, it will be remembered, was the case on the Japanese training-ship, the *Rinjo*, where only those who were unable to take the milk and meat provided succumbed to beri-beri. Or, all the conditions being equal, it must be accepted that the eight sick in the one cabin illustrated simply the common fact that all persons are not equally susceptible of any given disease; they were simply more sensitive than the others to the toxic agency.

In the occurrence of all the cases *on one side of the ship* is there anything so very remarkable?

There are, after all, only a given number of possible arrangements of a given number of objects and events. The twenty-eight Malays were divided into two parties, and of these the eight men attacked might have been either all on the port side or all on the starboard side, or distributed between the two, and what happened was the second of these three alternatives.

Finally, since clinical details are not supplied, it is permissible to doubt the diagnosis; for, as will appear immediately, Ellis has on occasion differed from colleagues of experience in his views as to what constitutes beri-beri; moreover, since these reputed cases were on shipboard, it is probable that they were examples of that commoner malady of sailors simulating beri-beri which, evidence will presently be submitted to show, is certainly not the same disorder.

Ellis has recorded an instance more interesting than this in the occurrence of beri-beri in a party of lunatics whom he specially isolated at the asylum in 1890, to be placed upon an entirely European diet. Reporting the result for the first time, *writing in 1903*,² he states: '*Thirteen years ago* I separated twenty healthy

¹ The fact that the galley was immediately next to the fore-castle of the affected men, suggests that the latter may have had, and taken advantage of, opportunities of getting larger rations of rice than their fellows. Were this so, the difference would be quite sufficient to determine the incidence of beri-beri.

² *Loc. cit.*

native patients on admission in two cottage blocks, giving them European food, and no rice whatever. At that time, though beri-beri was very prevalent among the natives, *we had never had any cases among Europeans or Eurasians*, and I wondered if in any way the food was in fault. My experiment had to be given up in less than three months, as more than half of the cases had by then developed symptoms of the disease. The opinion I then came to was that beri-beri was a place-disease, that the soil and buildings were infected, and that people dwelling there were liable to absorb the poison, whatever it was, that this absorbed in sufficient quantity was the cause of beri-beri, and this opinion I have never seen reason to change.'

Ellis offers no explanation of the persistent exemption of Europeans and Eurasians from the effects of this 'place-infection,' which, if it existed, they must have been continually absorbing equally with the other inmates.

This extract is from the *British Medical Journal* of November 14, 1903. In an earlier account of the same experiment (report on the asylum in Annual Medical Report for the Straits Settlements for 1903, p. 10, para. 8) Ellis prefaces his account with the remarks: 'Beri-beri has remained endemic in the asylum. . . . As to the cause of the disease, I regret being unable to report any progress. I am convinced, and have been for years, that the consumption of mouldy, microbic, or otherwise diseased rice is not the cause.' The account which appears in the journal is then given in identical terms, except that in the last sentence it is said: 'The experiment had to be given up in less than *six* months [not *three*, as above], as by then more than half the twenty cases had shown symptoms *more or less* of beri-beri.'

The importance of the conclusion to which his experiment thus led Ellis would seem to have been considered by him as of little value to others at that time, since he did not publish it until after so many years. But the experiment was at last given publicity, as the writer understands, to render the *coup de grâce* to the view of the dependence of beri-beri on rice, which the writer had then lately been urging locally upon the profession and the Government. It is thus put forth with absolute confidence on the part of the narrator that the result proves the conclusion which he submits—namely, that in the Singapore Lunatic Asylum beri-beri was due to some factor which was certainly not rice.

So important a conclusion demands the utmost scrutiny. It may be shown, both that certain fallacies of observation, certain

mistakes as to fact, may easily have accounted for the occurrence of beri-beri in the small separated party of persons fed on European food; and also, that it is more than a matter of doubt, whether the disease, the 'symptoms, more or less of beri-beri,' which Ellis, after the lapse of so many years, asserts to have appeared really indicated beri-beri or another malady.

Dealing with the first of these points, and assuming that the cases which happened were true beri-beri, was it absolutely certain that none of them got rice in any form?

In considering the case it is impossible to avoid the reflection that, in the East, it is one thing to order a particular diet to be used, and another thing to be sure that the order is strictly obeyed. In the case of natives, to whom abstention from rice is a sore privation, and who crave for the staple food they are accustomed to, whatever else is supplied, it cannot be doubted but that they would endeavour to secure rice whenever they could, and, where there were only native attendants, it is, to say the least, possible that they succeeded in getting it surreptitiously. But supposing, with Ellis, that they had no rice, that European diet only was eaten by these natives throughout the experiment, it remains to show that the flour in use was not itself principally made from rice, for the reputed wheat-flour of Eastern markets is often much adulterated.

If no rice were indirectly present, nor any derivative of it (such as glucose), it is to be supposed that, rice being certainly kept in the kitchens of the gaol, this was one of the cases where other aliments had become infected by contact with toxic rice, or places in which such rice had been stored, or in which the source of intoxication was other grain.

Such considerations, it seems to the writer, might quite adequately explain the occurrence of a few cases of beri-beri under the circumstances narrated by Ellis.

But again, it is possible also to challenge the accuracy of the diagnosis. Reference has already been made in this work to the complaint variously described by Lovell,¹ Macleod,² and others as 'acute anæmic dropsy' and 'epidemic œdema,' and to the confusion which has been made in more than one quarter between this disease and beri-beri. A great epidemic of it which happened in 1877-1880, and spread apparently from Assam, Shillong,

¹ *Indian Medical Gazette*, 1881, p. 342; 1882, p. 148.

² *Journal of the Epidemiological Society*, vol. xii., p. 55; *Indian Medical Gazette*, 1881, p. 148.

Calcutta, and other parts of India to Mauritius and the Seychelles, was referred to by Fayrer,¹ for instance, as beri-beri. It is probably a disorder widely distributed, forming the *mal de cœur des nègres* described a century ago, and prevailing largely all over the tropical belt, both of Africa and the East Indies, to-day. I see frequently sporadic cases of what appears to me to be this complaint in my own hospitals. Since the disease was differentiated by Lovell, Macleod, Davidson,² and O'Brien,³ it has been recognised as an entirely distinct disorder from beri-beri by such authorities as Scheube, Manson, and Cantlie; and those who will read the very clear and concordant accounts of it by these observers will find it difficult to understand why it should ever be confused with beri-beri. To the two affections there is, indeed, but the single symptom of dropsy common. Some digestive disturbance, some general malaise, some fever, even slight disorders of skin-sensation, there may be in either; but these signs are neither constant in both, nor in any way specific. But the differences are constant and very marked. An acute, abrupt fever always ushers in epidemic dropsy, which is never present in beri-beri. On the other hand, the prominent and often acutely painful disorders of sensation and movement, the tenderness and cramps of the muscles, the final wasting and paralysis, always present in some degree in every beri-beric, never occur in epidemic œdema.

This is a gulf of difference, and it might be thought impossible that the two diseases should be confounded. Yet, as already remarked, observers have confused the two disorders, and have thereby been led to erroneous inferences as to the causation of beri-beri.

Among the occasions upon which this mistake has been made it is highly probable that the experiment at the Singapore Lunatic Asylum was one. For one of the clearest and most succinct accounts of epidemic œdema has been given by Ellis, and is to be found in his Report on the Singapore Lunatic Asylum, included in the Annual Medical Report for the Straits Settlements for 1896 (p. 14, para. 21). Under the title of 'Anæmia with Anasarca,' he describes a disease as 'constantly prevalent since the new asylum was opened in 1887, . . .' and as being 'literally a scourge during 1895 and 1896.' His account of it, agreeing at all points with those given of epidemic dropsy by

¹ *Medical Times and Gazette*, June, 1880, p. 631.

² *Edinburgh Medical Journal*, August, 1881.

³ *Indian Medical Gazette*, 1879, p. 5.

other observers, makes it abundantly clear that the disorder was one and the same with the latter, and not beri-beri. 'Unlike beri-beri,' he says, in discussing the pathological position of the new disorder, 'there is no numbness, no tenderness of the calves of the legs or forearms, no huskiness of the voice, and no motor paralysis; neither does one ever see bullæ form or the skin slough. I have already referred to the consistency of the œdema' ['the initial œdema is not so firm as in beri-beri; it pits easily on pressure, commences most frequently in the lower extremities, but is often first noticed about the face and neck, or on the trunk']. 'The degeneration to be found in the peroneal, cutaneous, median, pneumogastric, recurrent laryngeal, phrenic, cardiac plexuses, and other nerves in those dying from severe beri-beri [and never, it may be added, absent in such cases] cannot be demonstrated in those dying of anæmia with anasarca.' He adds: 'Personally, I am strongly of opinion that the disease is beri-beri, although I am aware that my colleagues do not agree with me in this.'

There is thus a very explicit account of epidemic œdema as prevalent in the asylum at or about the period of Ellis's experiment. But there is not in this nor any subsequent report of his any definite account of cases such as would correspond with ordinary beri-beri as others know it. It must be concluded, therefore, that beri-beri was not then epidemic in the asylum, that the 'symptoms more or less of beri-beri' which the patients in Ellis's experiment showed were really ascribable to epidemic œdema, and hence that the conclusion which Ellis drew from his experiment against the rice theory was a mistaken one. As has been noted on a preceding page, Ellis appears to be veering round to this view himself.

Smart¹ has similarly been led to record a verdict against rice, because beri-beri attacked Filippino troops who 'drew flour in preference to rice for their rations.' It is incredible to the writer, whose experience of Malays of fifteen years' duration must lend his views some weight, that any body of natives bred on rice should voluntarily *entirely* give it up in favour of any other diet, or should do so for any length of time. In the absence of strict and convincing proof that no rice at all was taken by these troops, it may be doubted whether this is an instance of complete exclusion of rice from diet, and, if it were so, still the indirect sources of rice-intoxication remain as before to be excluded also.

¹ *Report Surgeon-General Army U.S.A., 1902-1903, p. 69.*

Writing in this sense, in 1905, to the Chief Surgeon of the Philippine Division, U.S.A. Army, the author was favoured by Assistant Surgeon-General Charles B. Byrne with the following reply :

'Concerning the matter of incidence of this disease among native troops' [statistics already quoted show that beri-beri has for many years been wholly confined to them], 'it is not improbable, I think, that your suggestion that the native does not always eat the exact ration drawn from the commissary is a proper one, and it may be that this failure to eat the bread-component issued, substituting therefor rice in sufficiency, accounts for the persistence of beri-beri among our native troops.'¹

I have, since this work was sent to press, come upon an earlier report by Ellis himself, which makes it plain, not only that the views which he held as to the definition of beri-beri, at the time of his experiment, differed from those of other authorities, but

¹ General Byrne courteously adds the following information, which is valuable as bringing the evidence derivable from the circumstances of the Filipino, which has already been touched upon, up to date :

'Certainly it [beri-beri] has very materially diminished, as shown by a comparison of the two excerpts sent herewith :

'EXTRACT FROM REPORT.

'April 11, 1905.

"*Beri-beri*.—This disease has been almost exclusively confined to native scouts. Reports from twenty of the principal scout posts in the department of Luzon show an admission rate of 43 per 1,000 of mean strength; the admission rates for the other two departments are unknown, but it is believed that they are practically the same as that above quoted. But one special report on beri-beri was received in this office; this report was forwarded to the Surgeon-General's Office under date of April 23, 1904.

"The general opinion of medical officers who have had experience with this disease is that it is a 'place disease,' and that by the removal of the patients from the infected locality the spread of the disease is arrested. A liberal dietary, with tonics and rest, seem to have produced the best results in treatment.

"Practically all the scout companies have been affected, and though the Chief Surgeon of the Department of Mindanao reports that the disease has occurred in a greater ratio than during the preceding year, the record of deaths from this disease shows but seven cases for 1904 as compared with twenty-three in 1903. The number discharged on surgeon's certificate of disability was three."

'EXTRACT FROM REPORT.

'June 30, 1905.

"The improvement in the matter of beri-beri has been very manifest; there is no note of any case among Americans, and the number of cases among the scouts is materially less than last year; there have been seven deaths from this cause, all occurring among native (scout) soldiers. The improvement is explicable as a result of the better ration given the native as a scout than he can command in civil life."

also that the illness which was then prevalent among his patients, and which (as one must infer from his account) the subjects of the feeding experiment also got, was not beri-beri, was not even any form of peripheral neuritis, as experts understand this malady, but what has been called 'epidemic œdema.'

The experiment itself is briefly described by Ellis in the Annual Medical Report for the Straits Settlements for 1902, p. 10 (and also in the *British Medical Journal*, November 14, 1903, vol. ii., p. 1268), in the following terms (see quotation on p. 405):

'Para. 8.—Beri-beri has remained endemic in the lunatic asylum throughout the year. . . . As to the cause of the disease, I regret being unable to report any progress. I am convinced, and have been for many years, that the consumption of mouldy, microbic, or otherwise diseased rice is not the cause. Thirteen years ago I separated twenty healthy native patients on admission in two cottage blocks, giving them European food and no rice whatever. At that time beri-beri was very prevalent amongst the natives (we have never had any cases in Europeans or Eurasians), and I wondered if in any way the food was in fault. My experiment had to be given up in less than six¹ months, as by then more than half the twenty cases had shown symptoms, more or less, of beri-beri. The opinion I then came to was that beri-beri was a place disease.'

In the Annual Medical Report of the same colony for 1896 the following appears in the report, also by Ellis, on the lunatic asylum:

'Anæmia with anasarca, constantly prevalent since the new asylum was opened in 1887, has been literally a scourge during 1895 and 1896, paralyzing the industries, increasing the death-rate, raising the rate of maintenance, and overworking the staff. . . . It is characterized by slight anæmia, considerable anasarca, and though endemic, not to say epidemic, is apparently neither infectious nor contagious. Occurring principally in the damp, low-lying, ill-ventilated wards of the lower levels, no part of the asylum is free from cases, and though the disease is most widespread during the rainy seasons, yet it crops up all the year round. European and Eurasian patients have been singularly free from the disease, and but two of the attendants (though these all live freely among the patients) have ever been attacked. Of the various agencies that have been assigned as causes, dampness, insufficient ventilation with inadequate air renewal,

¹ In the account in the *British Medical Journal*, 'in less than three months.'

insanitary hygienic surroundings, and malarial influences, are important. That malaria must be seriously considered in connection with the disease is, I think, indicated by the preliminary febrile state, the enlargement of the spleen, the masses of pigment frequently seen in the blood on microscopical examination, and the nature of the soil and height of the subsoil water of the site on which the lower blocks are built ; still it must not be forgotten that a large number of our patients have enlarged spleens on admission, that most of our cases dying from beri-beri have an enlarged spleen, and that quinine seems to have but little or no influence over the disease. It is difficult to say whether an incubation period exists. Some of the more intelligent patients complain of a feeling of lassitude and a disinclination for work or food for a day or so before the onset. The earliest symptoms are usually vomiting, occasional rigors, a feeling of coldness, headache, dry skin, temperature 101° to 103° F., and a soft quick pulse. The bowels, as a rule, are constipated, and the tongue thickly furred. The febrile condition is soon lost, but there appears to be no regularity about this, as sometimes it does not occur until after the anasarca has developed, and frequently there is no pyrexia whatever. The initial oedema is not so firm as that in beri-beri ; it pits easily on pressure, commences most frequently in the lower extremities, but is often first noticed about the face and neck or on the trunk, and rapidly becomes general. If the anasarca be excessive, and it often is, the patient lies helpless on the broad of his back somewhat resembling a drowned body, his condition being most pitiful. The amount of oedema is not necessarily a criterion of the gravity of the case, although when it is severe about the root of the neck and over the sternum there is usually a fatal termination. Ominous signs consist of embarrassed breathing from lung oedema, cardiac failure, hydrops pericardii, and continuous vomiting, the sufferer complaining of precordial pain and epigastric tightness. There is great restlessness, with distressed countenance, dusky face, and livid lips. The blood shows an appreciable loss of hæmoglobin, but little or no diminution of red corpuscles, the white corpuscles being in their normal ratio. The red cells, in a very large majority of cases, are seen to be markedly crenated, and pigmentary masses are numerous. The urine is scanty and high coloured, with a specific gravity ranging from 1.015 to 1.025, acid, with urates, and frequently phosphates in excess. Neither casts nor blood have ever been noticed, and albumen in only a few cases.

Functional cardiac murmurs and venous bruits in the neck are common. The knee-jerks are nearly invariably lost, the skin reflexes being unaffected. There is no paralysis, and the gait is normal. If the case is to terminate favourably, the anasarca slowly disappears, vomiting and other acute symptoms rapidly abate, and complete recovery takes place in from two to four weeks. Previous attacks appear to predispose to further attacks, for the same patients suffer again and again. New patients have been noticed to be especially liable to incur the disease. From time to time a case is noticed, having, with other symptoms, the gums spongy, deep red, or livid, and apt to bleed. It may be well to mention here that the asylum diet is liberal in fresh vegetables, and that all manner of diet has been tried as a prophylactic measure, but unfortunately without success. Death may be due to the water-logging of the lungs, but is more commonly due to heart failure, and is often sudden. The prognosis, though favourable, has to be guarded. As already stated, the amount of the œdema is no criterion of the gravity of the case; patients completely water-logged often make good recoveries if there be no heart or lung complications, while others who are but slightly swollen suddenly die from syncope. Post-mortem examination shows subcutaneous tissue throughout the body engorged with fluid, cerebro-spinal fluid in excess, lungs œdematous and congested at bases, hydrops pericardii (7 ounces to 1 pint), heart dilated and flabby, the right side filled with dark, easily broken-down clot, general venous engorgement, enlarged and congested spleen, congested and occasionally enlarged liver, enlarged and congested kidneys; the alimentary canal is usually normal and free from parasites, but *sometimes there is slight congestion of stomach and duodenum*. In former years renal cysts were frequently noticed, but latterly this has not been the case. Experience has taught that the most successful treatment is to immediately remove the patient to the driest and best ventilated part of the asylum (this varies with the wind), and to purge thoroughly and frequently with such drugs as elaterium, croton oil, calomel, and magnesium sulphide. Diuretics in combination with strychnine and iron (preferably an acid salt) are most valuable. In cases of heart failure nitro-glycerine has proved most serviceable. A decoction made from fresh pineapples is given freely to every patient, and certainly acts advantageously as a diuretic and possibly as an antiscorbutic. The diet should consist nearly entirely of milk, bread, and congee, any divergence from this

being usually accompanied with considerable gastric disturbance ; stimulants are indicated and of great use in severe cases.

' Various bacilli and cocci have been grown on gelatine and agar-agar from the blood of living patients, and after death from the blood, spleen, and elsewhere. Inoculations of agar-agar and gelatine tubes from blood have produced among other results an orange-coloured surface-growth which does not liquefy gelatine, and consists principally of micrococci and a fair number of short, very broad, and somewhat oval-shaped bacilli, with rounded ends. Attempts are now being made to obtain a pure cultivation of this bacillus with the object of experimenting with it on some of the lower animals. Cultivations from the blood, spleen, etc., post-mortem, have as yet given no useful result.

' From the above description the following questions arise : Is the disease beri-beri in a modified form ? Is it in any way due to malarial intoxication ? Or is it a new disease ? The presence of the ankylostoma duodenale has been suggested as a cause, but has been negatived. Personally, I am strongly of opinion that the disease is beri-beri, though I am aware that my colleagues do not agree with me in this. As in many cases of beri-beri, so in these cases there is slight anæmia, considerable anasarca, vomiting, a feeling of epigastric tightness, feeble onset, precordial pain, loss of patellar reflex, a flitting murmur to be heard over the area of the pulmonary valves, with, in serious cases, hydrops pericardii, œdema of lungs, and syncope. But, *unlike beri-beri, there is no numbness, no tenderness of the calves of the legs or forearms, no huskiness of the voice, and no motor paralysis, neither does one ever see bullæ form nor the skin slough.* I have already alluded to the difference in the consistency of the œdema. *The degeneration to be found in the peroneal, cutaneous, ulnar, median, pneumogastric, recurrent laryngeal, phrenic, cardiac plexuses, and other nerves, in those dying from severe beri-beri, cannot be demonstrated in those dying of anæmia with anasarca,* yet I have made out commencing degeneration in some branches from the solar and renal plexuses and from the inferior ganglion of the sympathetic in the neck in those dying of the latter disease. May not the disease be one affecting the sympathetic system alone, and thus differing from beri-beri only in the part attacked ? At any rate, more work will be done on these lines with a view of clearing up the matter, and in the meanwhile my state of mind on the subject is as that described by a well-known physician when he said : " When what is known is little and unsatisfactory,

and what is unknown is much and promising, then the tendency is almost inevitable to turn from the little that is known and make guesses about the greater unknown—and the guess is one's own, while the facts are anybody's; so the guess is fostered with a parent's love."'

This terse and clear-cut description of Ellis's is a cameo, a perfect picture of a disease which is not beri-beri.

A comparison of his account with the descriptions by Macleod, Lovell, Davidson, Manson, Cantlie, Scheube, and others of what has been variously termed by them 'epidemic œdema' or 'acute anæmic dropsy' will leave no doubt in any reader's mind that the latter complaint and Ellis's 'anæmia with anasarca' are one and the same malady. They agree at all points, and in nothing more emphatically than as to the complete absence from all, even the worst cases of epidemic œdema, of paralysis, or even of any definite signs of peripheral neuritis.

Between it and beri-beri there is indeed little more in common than dropsy. Moreover, this is not constant, being often conspicuously absent in true beri-beri, while in the other complaint, as Ellis himself has observed, it presents differences both in its nature and mode and place of origin. It remains to be added that there is no evidence in this report that any cases of true beri-beri were present in the asylum at this time. It must be supposed, therefore, that up to the date of this account, 1896, beri-beri, as it is understood by others, had not appeared in the asylum.

How, then, are the symptoms, 'more or less of beri-beri,' in the subjects of the feeding experiment narrated above to be accounted for?

Clearly the disease which these patients got was not beri-beri, but epidemic œdema.

3. **Scheube.**—An objection raised originally by Scheube,¹ since emphasized by Hirsch and others, and more recently repeated by himself, appears to rest on some misconception as to *facts*. In Brazil, Scheube believes, beri-beri bears no relation to rice. But his statement that rice is not eaten in Brazil, and that 'there has been no general diffusion of it [beri-beri] until recent times, while there has been no such change in the staple food of the inhabitants as would render its phenomenal outburst intelligible,' is certainly, in respect to the latter part of it at least, based on insufficient information.

¹ *Krankheiten der Wann. Land.*, p. 266.

It is true that it is only since 1860 that beri-beri has been fully recognised and become widespread in Brazil, but it is notable that it was just about this time (1858) that the principal ports of the country became open to world traffic, and that increasing facilities of steam communication everywhere made rice cheap enough to become adopted largely as an article of food.

The writer has been informed by a civil engineer, himself born in Brazil, and one of whose parents acquired beri-beri there, that the coincidence, if not the causal connection of beri-beri with rice was clearly recognised, his father having stated that it was from the beginning associated with the adoption by people of rice for their food. Moreover, Féris says,¹ Azevedo, who has studied the conditions of beri-beri in Brazil at first hand, expresses himself clearly in support of this view. The spread of beri-beri, and the use of rice, he says, have been everywhere in Brazil closely associated.

The original staples of food in South America, produced all over its vast region, are maize and manioc. In the Argentine wheat and barley are now also grown. Rice is cultivated only in Dutch Guiana and parts of Peru, Columbia and Ecuador, and not sufficiently for local needs.

During 1902, I find from official returns, no less than 89,375,310 kilos of rice were imported into Brazil, sufficient to supply 117,860 persons with 1½ pounds, or nearly 500,000 with 1 pound of rice *daily* for a whole year.²

It is not for want of rice that beri-beri should be lacking in Brazil, therefore; and there can be little doubt, it seems to the writer, that in that country, as elsewhere, rice and beri-beri have marched together.

From British Guiana an account of a small epidemic of beri-beri, investigated lately by Carter, comes pertinently to hand in the present connection. The cases observed were three. They occurred in a small party, dwelling in dense forest, whose food consisted of flour, salt fish, and *rice*.³

The accounts which the author has been able to peruse of the Brazilian malady leave him far from convinced as to the identity of the cases with East Indian (Malayan) beri-beri.

The caution seems to be worth repeating that not all epidemics

¹ *Arch. de Méd. Nav.*, June and July, 1881.

² *Board of Trade Journal*, 1903, vol. xl., p. 276.

³ Dr. R. Carter, *British Guiana Medical Annual*, 1902, also ref. in *British Medical Journal*, May 23, 1903.

of neuritis with or without oedema are of the same origin; and the determination of the fact that in Brazil such a form of neuritis, not ascribable to rice, occurs, cannot in any way affect the proved dependence upon rice of that form which is known as beri-beri.

4. **Baelz.**—The prevalence of beri-beri in Japan, Baelz¹ has stated, does not accord with *what ought to occur* if rice were its source. He asserts that it prevails more at the seaports and on the coast, where most fish is eaten, than in the interior, whereas, if rice be the cause, the contrary should be the case. Hirsch, again, and Scheube, have perpetuated this, as it seems to the writer, vague and loose objection, although it does not tally with Scheube's own account of the distribution, or Saneyoshi's, who would seem, as head of the Naval Medical Department in Japan, to be in a position to be acquainted with the facts.

Scheube says the disease first appeared at, and still infests, the capitals of Kyoto and Tokyo. The ports then became 'infected'—Yokohama, and later Hakodate—then there was spread along the western littoral to Nagasaki. 'A short stay in any one of these centres is sufficient to produce the disease.'²

5. **Ashmead.**³—More recently the same objection has been re-stated by Ashmead, who asks: 'If it [beri-beri] is due to eating certain sorts of rice, as the State surgeon of Negri Sembilan says it is, why did it not first appear at the ports of entry of such rice from China or Ceylon or India?'

The answer to all these objections is that in Japan, as elsewhere, rice causes beri-beri, not by infection, but by slow intoxication. The result depends not on mere contact, but, after its original intensity of toxic quality, on the quantity in which, and the time for which it is consumed. These conditions, again, depend on economic factors, the operations of which do not obey the same rules as contagion.

There is no reason why the disease should first appear at the ports of entry, for it is not necessarily in the ports to which products are consigned that they are consumed. The distribution of beri-beri, and even the season of its occurrence, obviously becomes largely—often perhaps entirely—dependent on economic conditions. Saneyoshi,⁴ reviewing the position in 1901, says of beri-beri in Japan that 'it was almost confined to a few decades ago to such populous towns as Tokyo, Kyoto, and sea-ports, as

¹ *Rutt d. D. ges. f. Nat. v. Volkerk. Ostas.*, Tokio, 1882, ix., 27, p. 295.

² Cited by Hirsch, *Handbook*, vol. ii., p. 422.

³ *Sei-i-Kwai*, August 31, 1903. ⁴ *Sei-i-Kwai*, April 30 and May 31, 1901.

Nagasaki, where *travellers crowded from every part of the country*, and had never prevailed over the whole land of the Empire *as is the case at present* (writer's italics). He explains how this crowding resulted in beri-beri in a very simple manner. In cities like Osaka and Tokyo it led to the number of persons boarding in other people's houses being increased. 'The reader will remember that *boarding-houses never use barley and rice, but rice only* for their boarders.'

Saneyoshi explains, as stated elsewhere, how fluctuations of beri-beri rose and fell with the price of commodities, dear rice involving smaller consumption of it, and local failure of crops entailing importation of cheap foreign sorts of rice. To the latter factor chiefly, he thinks, the general spread of beri-beri over the land was due. 'Besides, with the development of communications with foreign countries the importation of foreign rice, which is very inferior in quality, has given a plentiful supply to districts which have hitherto been short of rice.'¹ Moreover, he points out that the cultivation of barley—the second great staple of food in Japan—was largely becoming displaced everywhere by the more profitable culture of mulberries for the silk industry.

When prevalent among persons eating more proteid—fish—than others, if the observations represent facts, it is an indication that the rice eaten in such places is still relatively in excess, different in kind, or of worse quality. This might very well be the case on the coast as compared with inland districts, for the fisherfolk, not growing, would not use fresh rice, but would probably buy imported uncured white rice. They would, at all events, not be able to buy fresh rice, for such is seldom on the market, while the inland peasantry, growing their own staples, would naturally use fresh rice, and probably dilute it with barley, too.

The observations of Saneyoshi throw light upon another dark place in beri-beri epidemiology—viz., the greater incidence of the disease, noted by both Azevedo in Brazil and by Simmons,² Scheube, and others in Japan, upon the relatively *wealthy* as compared with the poorest classes. Rice, always dearer than barley in Japan, and much more so than corn and manioc, the native staples of food in Brazil, *is naturally eaten less by the poor*. The rich, in this as in other cases, thus pay dearly for luxury.

¹ The writer's explanation of the same facts would be that stale rice was thus substituted for the fresh, or less stale rice of the country.

² *Med. Rep. Nup. Mar. Cust. China*, 1880.

6. **Stanley.**—Stanley,¹ who studied the conditions of beri-beri in Shanghai, and whose observations as to fact entirely accord with everything expressed in this work, finds himself obliged to deny a causal action to rice, on the ground that an experimental change in the quality of rice supplied to prisoners during two months made no clear difference in the resulting beri-beri. A fall in the case-rate occurred, but this coinciding with the period at which the disease was usually found to decline in the gaols, Stanley esteemed it independent of the change of diet.

'Inasmuch as,' he says, 'apart from rice the food supplied to three out of four prisons, at all of which the disease was about equally prevalent, was obtained from different sources, and a change of rice for all prisons to one of recognised good quality produced no well-marked effect on the prevalence of the disease in two months, food-infection would appear *not* to be a factor.'

This objection is disposed of by considerations already adduced, but which may be reiterated. Rice of 'recognised good quality' may be as toxic as any other—is, indeed, it has been shown in regard to some varieties, likely to be even more so.

Had Stanley's experiment been conducted with another grain instead of rice, with *fresh* instead of *stale*, *red* instead of *white*, or with *cured* instead of the *uncured* variety, the result, one cannot doubt, would have been to diminish if not abolish the disease. But, as it seems, one uncured white rice was substituted for another.

7. **Fiebig.**—The remarkable objection, which his high expert authority renders it impossible to dismiss lightly, has been lodged against the rice theory by Fiebig—that in the Moluccas beri-beri freely occurs among natives who yet eat no rice, but use sago for their staple food. In his important paper on 'Beri-beri among the Natives of the East Indies'² this author shows that a disease identified with beri-beri had been recorded as occurring from quite remote times among almost all the islands of this group.

Thus, in *Amboina* Valentin had noted it in 1724, Epp in 1844, and it had been mentioned in official reports in 1889.

In *Saparoea* a considerable epidemic had been noted in 1844 by Robinow, and cases had been seen by Heijman. In *Banda*, according to Swaving, the disease had followed the arrival of Governor-General Both there in 1611. In 1874 it was noted as

¹ *Journal of Hygiene*, July 1, 1902.

² *Gen. Tijdschr. v. Ned.-Ind.*, Deel xxx., Nos. 4, 5, pp. 463-504.

happening sporadically among natives by Van Hengel, and was epidemic in the gaols and barracks.

At *Batjan* Bernelot-Moens had observed beri-beri to affect especially the sago-workers, and had attributed it to the fact of their sleeping on the swampy ground among the sago-palms. 'It is known,' says Fiebig, 'that the natives of the Moluccas *plant* no rice, but live upon sago.' This fact entirely does away, for me, with the theory that beri-beri depends upon a poison *peculiar* to rice' (p. 465).

Certainly, if the Amboinese, never eating any rice, gets beri-beri, Fiebig's deduction, that beri-beri comes not from rice alone, must be accepted. The main contention of this book is, however, to establish rice, not as the sole possible, but as one, and that the usual, medium of the poison which causes this disease, a position in no wise invalidated by Fiebig's observation.

But that observation and the conclusion to which it leads are, as it would seem to the writer, either so important that they should have been presented in a mode less open to criticism, or the evidence upon which they are based is so meagre and unsure that they may be taken as of little importance.

For what is the evidence as to the disease being *beri-beri*? Records for the most part of great antiquity, not given in detail, so that the possibility, always also a probability, that the disorder may have been in reality something else—e.g., *epidemic œdema*—cannot now be properly estimated.

What is the evidence for the belief that no rice was *eaten* by these islanders? Merely the presumption afforded by the fact that they did not *grow* rice. But rice can be imported.

Fiebig supports the extra-rice derivation of beri-beri thus suggested by reference to the Ainos, the aborigines of Yezo, whom he quotes Scheube as asserting to acquire beri-beri while subsisting, as they usually do, in their free state on such things as fruit and roots, game, fish, and millet. 'They get rice by barter from the Japanese, only to make an intoxicating liquor from it' (*loc. cit.*).

But Grimm,¹ who also studied these aborigines at first hand—this we have already cited—attributes the beri-beri which the Ainos incur to their adoption of Japanese habits of feeding—to the use of rice—in preference to their own. And he expressly states that, so long as they preserve their native customs in this respect, they do not get beri-beri.

¹ *Klin. Beob. ii. B.-B.*, Berlin, 1897.

Fiebig further adduces in support of the sago factor a Report by the Resident of Rieuw, Halewijn, dated April 19, 1888 (*loc. cit.*, p. 483):

'Beri-beri occurs among the native inhabitants of the districts of Lingga and Karimon, and in the capital, Tandjong Pinang, and at times in the district prison, and on the Government steamer whenever the latter returns from a trip to Karimon or Lingga.' Fiebig draws attention to this as evidential of place-influence.

'Agriculturists, fisher-folk, and artisans, all perfectly free, were attacked, whether they lived in houses or lived in sampans (boats).

'Some ate sago, others rice, vegetables, and also fish.'

'In the Karimon district most cases happened among the sago-workers'—were not, perchance, the sago-workers the most numerous?—'who had to spend long hours of labour in the water and the mud.'

'The Karimon people assign as the cause of it working among the sago-palms and cleansing the sago.

'The mortality is not great; recovery, as it seems, complete.'

The last sentence hardly conforms with, at all events, bad cases of beri-beri.

This was written in 1890, but Dr. Fiebig would, doubtless, have collated with the foregoing the fact, noted by Grijns later,¹ that fowls fed exclusively upon sago also may develop polyneuritis similar to that produced by stale white rice.

In all this it seems to the writer there is no definite proof of the existence of beri-beri, still less unequivocal proof of its dependence on sources other than rice. But the facts offered are suggestive, and everything tending to elucidate the etiology of beri-beri so important, that it may be hoped these interesting statements may at some time be made the subject of further inquiry.

From the point of view of an objection to the rice theory formulated in this book, however, it is clear that Fiebig's observations, if they prove something for sago as a possible cause of beri-beri, at least in no way oppose rice as its common and chief, and probably sole, source.

8. **Durham.**—Durham,² as the result of observations made at Christmas Island, has specified several difficulties in the way of

¹ *Med. Lab. Weltevreden*, 1900.

² *Journal of Hygiene*, January 1904.

accepting rice as the beri-beri agent, which, however, are but iterations of old ones, and offer no new features.

(1) One of these was, that 'all the rice-eaters obtained rice of the same quality from the same store. Yet it was the coolie who suffered.'

The explanation of which is that, as Durham himself carefully explains earlier in his article, 'the coolie' had an actually different ration. 'Beri-beri was practically limited to the coolies, *who alone were subsistent on the contract diet*. The only other persons affected among the Chinese workers of the company were a few of the *tukangs*' (mechanics). 'The *tukangs* obtained their own victuals, while the coolies live on the contractor's supply, which in the case of the *laukhehs* may be added to by their private means, especially pork and pigeons.' Unfortunately, Durham does not give the actual 'contract' fare on Christmas Island, or the sort of rice used. Doubtless, the diet of the *sinkhehs*, there as elsewhere, consisted chiefly of ordinary white uncured rice,¹ some vegetables, and fish, with rare additions of fresh meat. In fact, the Chinese contract coolies on Christmas Island, like those in Malaya, as compared with others, ate white stale uncured rice, and ate it in excess.

(2) 'There was a severe outbreak of the disease amongst a party of *Tamils*. This was not coincident with any recrudescence amongst the Chinese coolies, who lived about a mile away. These *Tamils* ate in company with the Malays, the accessory foods were dealt with by each, but *the rice was taken actually out of the same pot of cooked rice*. Why, then, should not the Malays also have suffered, if the rice were at fault ?'

The exact answer to this question depends on circumstances which are not disclosed ; but several possible explanations are obvious, as (a) that the rice was not shared by the two races for equal lengths of time — it takes usually several months, and always many days, to get beri-beri ; (b) that, though the same rice was used, it was not taken by both races in the same quantity, either absolutely, or relatively to the rest of the diet. The accessories which were 'dealt with by each' separately may have made all the difference. A little fat, a little proteid, more or less, makes all the difference between getting the disease or remaining healthy. Enough examples of this have been given. Those who know the Tamil will recognise how small the prob-

¹ I have since been informed that not only is this so, but the coolies have got, what they get nowhere else, *i.e.*, quantities *ad lib.*

ability would be that he should supplement the rice given him by any accessories for which he himself should have to pay. Those who know the Malay will be aware that nothing but want of means would prevent his buying for himself any extra articles of food which he desired.

(3) That 'in the Pudooh Gaol patients in the gaol hospital were recovering, whilst about the cells or work-places their mates were being invalidated day by day. They ate of the same rice, which was all cooked together.' It seems sufficient to repeat this objection for its hollowness to appear. It does not follow—is, indeed, unlikely—that the sick and the healthy consumed the rice in equal degree. Moreover, the natural tendency of the system, under any poisoning, is to recovery. In this effort there is at times success. As in the case of alcohol and tobacco, to take familiar examples, some degree of toleration, if only temporary, is established, which enables the stricken to survive the effects and to endure the continued application of poisons, even in doses which, if given initially, might have proved lethal.

(4) 'When the distribution of the disease throughout the world is included, rice has also to be put on one side. Thus, in the outbreak at Richmond Asylum, Dublin, as on many Norwegian ships, it is stated that no rice had been consumed.'

So far as the writer is acquainted with them, the chief peculiarities in the distribution of beri-beri have already been alluded to and explained in these pages.

And it will probably be conceded that in every part of the world rice has been shown to be at fault.

The validity of Durham's objection rests on two assumptions: (1) That the beri-beri so called of the Richmond Asylum and of other places in Europe and elsewhere, the ship-epidemics and the beri-beri of Christmas Island and the tropics generally, are clinically the same—*i.e.*, that they are an identical disease—and (2) that all disease manifestations clinically identical must own the same single cause. Can either of these postulates be proved?

It may be shown that, so far from the asserted beri-beri epidemics of temperate climates being clinically exactly the same, there are sufficient differences in the symptoms exhibited in the instances reported to raise great doubt as to that identity, or at all events to justify some of these outbreaks being regarded as at most but modified examples of the usual beri-beri effect. Again, that all identical clinical conditions must own a single

specific cause is an assumption which the facts so far determined about polyneuritis certainly do not justify. If the polyneuritic asylum epidemic and other epidemics of Dublin and elsewhere, therefore, be proved identical (clinically) with tropical beri-beri, and if rice be excluded from their causation, no more is proved thereby than the fact, otherwise not new, that extensive 'peripheral neuritis' may be due to more things than one.

While Durham's assertion, then, must fail, as an objection to our theory, it leads naturally to consideration of the questions, of very great interest and importance in themselves: Are the asylum epidemics of temperate climates and the numerous ship-epidemics true 'tropical' beri-beri? And, if so, can they be shown in any way to depend on rice?

Beri-beri outside the Tropics.—'*Beri-beri of Temperate Climates—Ship Beri-beri.*'—For obvious reasons the many questions involved in the possible etiology of recorded European and American epidemics of peripheral neuritis, or ascending myelitis, into which (nosologically or descriptively) they merge, cannot be more than touched upon in this work, primarily concerned with the demonstration of the common source of the tropical malady. But it is proposed to submit material sufficient to indicate the probability of the following several propositions:

(1) The peripheral neuritis occurring in great epidemics on land, outside the tropics, in asylums, and elsewhere, is not identical with tropical beri-beri—though it may be a modified form of it, the result of action of the same agent possibly, under circumstances modifying the latter's activity.

(2) Ship beri-beri bears a similar relation to both of these.

(3) Rice, the medium of conveyance of the poison in tropical beri-beri, cannot be considered excluded from the causation of other epidemics merely because it has been stated not to have been directly used as food. There are indirect ways in which rice can come into consumption.

(4) Supposing rice, eaten directly or indirectly, to have been excluded, yet the agent to which the toxic quality of rice is due, or some variety of that species of agent, may be conceived to be the cause of the epidemics of the modified disease, conveyed through other grains—possibly through entirely different sorts of food.

The asserted beri-beri of temperate climates, between which and rice no connection has been traced, certainly presents a

difficulty; but that difficulty does not, however, affect the question of beri-beri as a *grain-intoxication*, nor the proofs which have been given of the dependence of beri-beri upon rice elsewhere.

That relationship, having once been completely established, as those who have weighed the evidence submitted in this work will admit, cannot be affected by the production of even identical clinical results in other places through other causes. The issue becomes rather whether the term 'beri-beri' can be properly applied to conditions in which the operation, neither of *rice* nor of the rice-poisoning factor, can be traced. In view of the numerous causes to which conditions of peripheral neuritis, not essentially different from beri-beri clinically, have been assigned, it may be said, to anticipate a later argument, to be a matter of little moment if one more be added to the group.

Other Causes of Peripheral Neuritis.—*Oppenheim*¹ gives the following causes of multiple peripheral neuritis: Alcohol, arsenic, lead, copper, carbon monoxide, anilin, ptomaines, leucomaines, the puerperium, auto-intoxication, 'obstipation,' diabetes mellitus, icterus, enterica, variola, scarlatina, influenza, erysipelas, pneumonia, acute articular rheumatism, parotitis, gonorrhœa, diphtheria, septicæmia, hepatic cirrhosis, syphilis, malaria, dysentery, tuberculosis. Besides all these, he observes, there are the 'idiopathic' cases, of which the source cannot be detected.

*Gowers*² mentions as yet other causes—diarrhœa, and measles—which latter disease both an acute ascending paralysis and localized poliomyelitis have been observed to follow.

*Ross*³ has specified among diseases gout, and among toxic agents carbon bisulphide and roburite, as leading to polyneuritis.

To these should be added chancroid, a large number of cases of which are described by *Maitland*.⁴

Oppenheim and *Gowers* both take alcoholic neuritis as the type of a process, the clinical results of which in identical form may be reproduced as the direct sequel or effect of any one of the antecedents above named.

*Bury*⁵ appears to hold the same view, but to find convenience

¹ 'Diseases of the Nervous System,' trans. E. Mayer, second annual edition, 1904.

² 'Diseases of the Nervous System,' 1893.

³ *The Medical Chronicle*, October, 1890.

⁴ *British Medical Journal*, February 4, 1899.

⁵ Allbutt's 'System of Medicine,' vol. vi., p. 673.

in classifying forms according to the predominance of the motor or sensory symptoms noted.

In the conception of multiple peripheral neuritis framed by these authorities we have therefore a single result—an identical clinical condition, accepted as the direct effect of some thirty or more specific and distinct, even widely different, causes.

It is possible that this view may be quite wrong, that all cases of polyneuritis at present ascribed to so many agents may really be due to a single specific and hitherto unrecognised cause, the operation of which is merely favoured, or perhaps only rendered possible, in the presence of the factors hitherto supposed causal.

The agent must necessarily be conceived as widely distributed, and almost constantly able to act, when the favouring condition presents itself, and this condition could be fulfilled by such a factor as a common form of poison in grain might be.

Without, however, pausing to discuss further the probabilities of these two conceptions, the point may be insisted upon that rice, or other grain, as a cause of polyneuritis, is but a single one among so many recognised effective causes of a precisely similar condition. Their number is not limited by enumeration. It is not only possible, but probable, that more of equal validity will be added to the list. Therefore, not every epidemic of polyneuritis, resembling beri-beri as a polyneuritis must do, for which none of the other recognised causes can be assigned, need be identified with beri-beri, for the undiscovered sources of the condition may be as numerous as those that are known. The argument drawn from instances of beri-beri, so described by pure assumption, against rice as at any time the cause of the disorder elsewhere, because in the assumed cases no rice was used, or traceable, is thus a glaring fallacy.

On the one hand, since *beri-beri*, as seen in the places of its most frequent occurrence, has been clearly shown to depend upon a poison, of which rice is usually, and its derivatives less commonly may be, the vehicles, the term should be reserved for those cases and epidemics only, in which the action of this agent is made certain, or at least shown to be probable. *Cases in which the rice-agent can be definitely excluded*, however closely they may conform to the type, *have no title to the term 'beri-beri.'*

But if, on the other hand, the term 'beri-beri' is to be extended to all cases clinically conforming to the type dependent on rice, under the conviction that they are, in their pathogenesis as well as symptoms, the same, some other mode of access of the

rice-poisoning agent has to be sought. Durham, although he will not accept the conclusion to which they logically lead, puts these issues clearly when he goes on to say: 'It would be necessary, therefore, to bring such outbreaks [of beri-beri in temperate climates when rice was not involved] into line with the rice theory, either by supposing that it was not the same disease, or else that other cereals may be similarly infected with the causative agent.'¹

This, the logical conclusion, which whoever will weigh all the evidence must adopt, is the position taken up in this book, the view that beri-beri is a grain-intoxication.

From this point of view it can in no way either help or impair the proof of the dependence of beri-beri, as it is known in the tropics, upon rice, to discredit the diagnosis as beri-beri of cases of peripheral neuritis occurring in temperate climates and on ships, under circumstances where rice appears to play no part. Yet it is remarkable that epidemics of such cases occurring on land, at least, are very few; and perhaps more so, that they usually present such points of difference that even experienced observers may be divided in opinion as to whether they are 'true beri-beri.'

Among such epidemics, which received much attention at the time, and were carefully described, were those occurring at the Richmond Asylum, Dublin, at the Alabama Bryce Insane Hospital, Tuscaloosa, U.S.A., and at St. Helena.

Richmond Asylum Epidemy.—Of the former epidemy I extract the following from the several accounts given by Norman, the superintendent.²

The cases occurred first in June, 1894, and continued to do so until September, declining in October, and ceased in December.

In 1895 there were no cases.

In 1896 a second epidemy began in July, lasting till the end of the year.

¹ He adds: 'That is to say, we reach the region of pure speculation without solid facts in support.' But such speculation need not therefore be idle. Not seldom has it happened, that the speculation, the theory, which, like a lantern, directs search in the likeliest quarters, has been the means of the discovery of the facts. The latest, and most brilliant of such examples was, perhaps, the induction of Manson, which was the 'onlie true begetter' of the discovery of Ross's malaria-mosquito cycle. The causes of pellagra, of gout, of scurvy, to seek no more examples, remain in this sense speculations, yet the views which are accepted as to the causation of those diseases are not on that account condemned. The theory of gravitation is yet a 'pure speculation.'

² 'On Beri-beri in Temperate Climates,' *British Medical Journal*, September 24, 1898, ii., p. 273; also 'Editorial,' *British Medical Journal*, 1897, ii., p. 168.

In 1897 a third epidemic began in July, when most of the cases happened. There were many in August, and a few scattered over the year till its end. The figures were :

Year.	Daily Average Number of Patients.	Cases of Beri-beri.		Deaths from Beri-beri.		Total Cases.
		Males.	Females.	Males.	Females.	
1894	1,503	127	47	18	7	174
1895	?	nil	nil	nil	nil	nil
1896	1,686	31	76	20	6	114
1897	1,800	47	199	3	8	246
Total	—	205	322	41	21	534

The case-mortality for the whole series was thus at the rate of 116 per 1,000.

In 1894 none of the staff were attacked, but in 1896 seven nurses, in 1897 six nurses and two attendants suffered.

The first condition usually noted was pretibial œdema, and tenderness on deep pressure over the calves was early noticeable. A certain amount of superficial anæsthesia could be made out. There was 'unconformability' of heart-beat and pulse at the wrist, tachycardia, vomiting in 5 per cent. of the cases, and sometimes diarrhœa, rarely both together.

Among the motor signs the first most remarkable was loss of power in the peronei and foot flexors. 'There was almost invariably a tendency to drop and invert the foot. Actual wrist drop occurred in less than 1 per cent. of all our cases, and was only extreme in one case. . . . Tendency to contractions appeared in half a dozen cases. . . . Only two patients remained crippled from this condition. The most frequent spontaneous complaint was of pain as if insects were creeping over a sore surface—"painful formication," the *pirri-pirri*¹ of the Japanese.'

Patches of hypo- surrounded by hyper-æsthesia were often noticed. Anæsthesia, fugaceous, as Scheube describes, was 'in

¹ I believe it has occurred to no one to suggest that in this word we may have the original of 'beri-beri.' As an almost constant feature in the complaint as described by Japanese, who have suffered from epidemics of it from very ancient days, the word is likely to have got spread abroad all through the East, over which the race sailed and traded extensively in days before Europeans ventured in Eastern waters.

some cases often noted. The superficial nerves were tender, sometimes extremely so.' In the few cases in which test was made for anæsthesia of the pharynx it was 'always present.' There was œdema of the *fundus oculi* in some, and perhaps amblyopia.

'In one case there was unilateral paralysis of the third nerve, ptosis, external strabismus, and dilated pupil. Engagement of the third nerve has rarely, if ever, before been observed in beri-beri.' The onset was extremely insidious—weariness of legs, with occasional cramps, often preceded more definite signs by a considerable time. The course was extremely variable. Norman notes the occurrence of 'sudden changes for the worse, and the extreme frequency of relapse.'

'There were remarkable differences between the three epidemics. In the first there was high case-mortality, *shiyo-shin* [cardiac angina] was common, there was a large number of cases of general well-marked œdema, and motor paralysis was pronounced. In the second case-mortality was lower, *shiyo-shin* rare, general anasarca was infrequent, motor troubles less marked. In the third the great majority of cases were of the type which Scheube calls "rudimentary," but there was quite a special tendency to the occurrence of *pirri-pirri*. Yet in each of the later epidemics there were often cases recalling the condition prevalent in the first.'

'In the epidemic of 1894 males chiefly were attacked, in that of 1896 this order was reversed.' The *British Medical Journal* reports the following further observations of Norman, dealing chiefly with the epidemic of 1897:

'A great number of the cases have exhibited a peculiar thickening of the tissues in the lower third of the leg resembling solid œdema, not pitting, or pitting very slightly, as one discovered on attempting to pinch up the skin. Dr. Conolly Norman states that he found a similar condition in ordinary alcoholic neuritis. The motor signs have not been very well marked. The relaxation of the knee- and ankle-joints is extreme in most cases, and is present probably in all. In many this condition permits the legs and feet to be placed in positions resembling those of subluxation. This condition gives rise to a characteristic wobbling gait at the knee which the Japanese designate, according to Professor Anderson, *gaku-gaku*.'

This epidemic at Dublin naturally attracted great attention, and the cases were seen by many experts, among whom there was,

as the writer has been given to understand, diversity of opinion as to the proper diagnosis.

While the cases agreed at many, even most, points with the classical type of beri-beri as seen in the tropics, as, indeed, with all forms of peripheral neuritis, there were also many differentiating features, some things absent which are not usually so, some things frequently present or conspicuous which are rarely so in tropical beri-beri.

The patches of hypo- surrounded by hyper-æsthesia (*anæsthesia dolorosa*), the contractions, the excessive joint-relaxation, the cranial implications (*ptosis, œdema fundi*), the 'solid œdema,' are among the latter class. Among the former, the mildness or infrequency of paresis, or paralysis, the complete absence of tetraplegia, of paraplegia, of even bladder paresis—one of the commonest beri-beric signs—the infrequency of great anasarca, or of pain and distress about the epigastrium, often severe in true beri-beri, or ascites, sudden onset and reduction of œdema, above all, the absence of the atrophy invariably present and rapidly following on the paresis, and usually a most marked sign of the disease.

Among 534 cases of beri-beri of the tropical type sufficiently severe to be fatal to 62, it may safely be said that there would not fail to be some of profound and almost complete and rapid flaccid paralysis like that of Landry's disease, and many who would be nearly paraplegic would be observed. There would be sudden and enormous anasarca, as rapidly subsiding in others; and in all, even the mildest cases, the ensuing atrophy would proclaim the direct injury done to nerve endings and muscles.

Clinically, therefore, so far as can be judged by the descriptions given, the Dublin cases do not accord with the type of tropical beri-beri. Nor, it seems, is there any other good reason why they should be classed particularly under that name. The asylum, the *British Medical Journal* states, long overcrowded, was infested by other diseases. 'Tubercle was unduly prevalent. . . . Other zymotics were rarely absent. . . . Dysentery, once a very prevalent disorder in Ireland, happily rare in modern asylums, seems always to have remained prevalent in the Richmond Asylum.' Dysentery, according to Daniels, is a frequent cause of neuritis.

'The inmates of the asylum get no food which is not in common use in the city of Dublin generally.'

The ordinary diet consisted of :

Bread, $1\frac{1}{2}$ pounds.

Meat, 8 ounces (men)—five days fresh beef, once
once bacon—or 7 ounces (women).

Potatoes, 1 pound.

Vegetables, 1 pound.

Tea, 1 pint at breakfast.

Cocoa, 1 pint at supper.

'Rice was little used, practically only as a medical diet. It being suggested after the epidemic that there was deficiency of fat, butter was afterwards specially added to the diet, but without effect in preventing the later epidemics, however.

Epidemy at the Alabama Bryce Insane Hospital, Tallapoosa, U.S.A.—At this institution, Bondurant¹ says, 'during the ten years ending February, 1895, there occurred not more than a dozen cases of multiple neuritis, and all of these, with two exceptions, were of the alcoholic or syphilitic variety. The exceptions were due, one to an aneurism, the other to trauma. Between February, 1895, and February, 1896, thirteen other cases happened in a hospital population of about 1,200, for none of which could any definite cause be assigned. Three died.

Bondurant describes the thirteen cases carefully in detail, and compares, but does not (in 1896) identify, them with beri-beri.

'A fairly representative composite clinical picture drawn from the cases we have seen (and which I abbreviate) shows the following symptoms as prominent :

'After a period of malaise, lasting from a few hours to several days, with the discomfort common in the prodromal stage of acute disease, sometimes with a distinct chill, usually with a rise of temperature, together with anorexia, dryness of mouth, sore throat, furred tongue, offensive breath, and constipation, the patient begins complaining of pain in the affected part, the most often . . .

'Muscular weakness and *inco-ordination* is then noticed; the deep reflexes are also abolished (*noted in all our cases*). The muscular weakness increases in severity, *the gait becomes ataxic*, patient becomes unable to walk up and down stairs; *unable to stand with eyes closed* [Romberg's sign]; 'the weakness may become only a paresis, but in many cases there is al

¹ 'Report of Thirteen Cases of Multiple Neuritis,' etc., *Medical News* (New York), October 3, 1896.

complete paralysis at the height of the attack, with the characteristic wrist-drop or foot-drop. . . . Trophic derangements . . . are also *frequent*, as œdema, eruptions, sores, discoloration, etc., of the skin, or, more rarely, herpetic eruption. . . . When the motor paralysis becomes marked, there is always atrophy. . . . Contractures may result.' (Bondurant draws attention to certain resemblances to and differences from neuritic pseudotabes.) 'The symptoms common to the two diseases are: The pains in the legs, the sensory perversions, *muscular inco-ordination*, *ataxic gait*, *swaying with closed eyes*, absence of tendon-reflexes.

'The differences are: The girdle-pain of tabes is not present in neuritis, etc. . . .

'An examination of about three-fourths of the cases was made after the acute stage was passed, without discovering any indication of increased renal secretion. . . .'

The early hypermyotonus, almost invariable, the early and continued cardiac derangement, also almost invariable, the œdema, beginning first in scrotum or upon the shin, rarely absent in any, and never absent from a *group* of cases of beri-beri; the severe cardiac and respiratory crises, the phrenic paralysis, which are such common events—the absence of these, and the presence (in almost all the cases) of *ataxia* and Romberg's sign, which my experience cannot permit me to describe as symptoms of beri-beri—all these differences make it quite certain for the present writer that, whatever this epidemic may have been, it was not beri-beri. Nor, as we have seen, did Bondurant in 1896 so describe it.

'After a period of immunity the disease reappeared in the late summer of 1896, when, following a season of unusual dryness, . . . 58 patients were attacked, 53 of these cases occurring during September and 5 during October.' Whites and coloured were alike attacked. There were in all 71 cases in a population of 1,200.

'While the negroes suffered in smaller numerical proportion than did the white patients, the disease in them assumed a severer form, 6 out of the 7 cases terminating fatally.' Of 64 whites affected, 15 died. The epileptics suffered most. Of 82 such patients, 32 had the disease.

Bondurant, after this outbreak, concluded the disease to be identical with beri-beri, and so described it.¹

¹ 'Endemic Multiple Neuritis (Beri-beri),' etc., *New York Medical Gazette*, November 20 and 27, 1897, vol. lxvi., pp. 21, 22.

In the second epidemic there were more acute cases. Many resembled, were 'typical examples,' Bondurant says, 'of beri-beri hydrops,' a form not seen during the first and milder outbreak.

Nearly all the cases had fever, some considerable pyrexia.

'About 40 of the cases which occurred in 1896 showed the heart symptoms very typically.' These had tachycardia, continuing long after other signs were remitting, dyspnoea, venous pulsation in neck, bruits, occasional cyanosis. 'Fourteen of the twenty-one fatal cases died directly of cardiac failure.'

One or two cases died of a very acute and rapid attack resembling pernicious beri-beri. The typical case cited as characteristic of this condition died in seventy hours from onset of first symptom. He had—I commend this to Dr. H. Wright—'no gastric disorder.' Gastro-intestinal disturbance was a prominent feature, however, in most of the cases.

'The temperature was in about half of the cases elevated in the beginning, but usually subsided to normal within a week or so. . . .

'In most of the febrile cases, as well as in most of these in which the neuritic symptoms were severe, there was marked disturbance of the digestive apparatus, consisting in anorexia, nausea, offensive breath, raw red-edged tongue, with, in some cases, gaseous distension of the intestines and much tenderness on pressure over the abdomen. Constipation was the rule, though in one or two cases there was diarrhoea. In some instances the gastro-intestinal disorder formed the most prominent feature of the case, quite overshadowing the local neuritis. These cases resembled typhoid fever minus the typhoid temperature. . . . Neither the fever nor the gastro-intestinal symptoms were seemingly essential features of the disease, since cases exhibiting all of the milder nerve-symptoms remained free from systemic disorder throughout the course of the disease.'

None of the scorbutic gum appearance so common in Malaya, none of the faucial redness to which Bentley and Durham refer, in any of these cases.

In the case which Bondurant selects as a 'typical beri-beri hydrops' there was renal implication, 'urine scanty, high-coloured, and albuminous.'

'Many of the patients had suffered from mild chronic forms of renal disorder, and in a small proportion of them albumen and casts existed in the urine in some quantity. In, say, one-half

of the cases—the milder half—no exacerbation of renal disease nor development of any perceptible degree of renal inadequacy was noted. In the other half—including the severe cases and those in which there was fever or gastro-intestinal disturbance—there was some evidence of renal irritation or disease, shown by the appearance of albumin or increase in its amount, together with increase in the number of casts. These abnormalities in the course of a few weeks returned to their former state, or disappeared entirely.’

‘Fluid in the pericardial sac was not diagnosed during life, nor found in any one of the seven fatal cases subjected to post-mortem examination,’ a contrast with the findings usual in beri-beri in the tropics.¹

The œdema ‘*always* appearing in true beri-beri,’ Manson says, ‘first beneath the skin over the crest of the tibia,’ in Bondurant’s cases was oftenest ‘of the ankles and legs. . . . It began in the *feet* and extended upwards.’

Early hypermyotonus is again not mentioned. Apparent hypertrophy of the tender muscles, also commonly seen in early beri-beri, is not noted. Scrotal œdema (which, when it appears alone, without renal disease, is so characteristic of beri-beri) and bladder-paresis—a frequent trouble—are not mentioned.

There is no mention of tender nerve-trunks, girdle-pains about the base of chest or round the neck, which, *pace* Bondurant’s remark cited anteriorly, have been noted from historic times as peculiar to beri-beri, also find no place.

Nothing is said by Bondurant in this account as to the diet of the patients, so that it is quite possible *rice* was used. But this point need not be insisted upon here. What is submitted for consideration is the divergence of the cases from the tropical type ; for it can safely be said that no group of cases so numerous and so fatal occurring in the tropics would have presented the same picture. If this were beri-beri, it was certainly beri-beri greatly modified.

The active toxic factor is ascribed in this instance, with very convincing reasons, to something in the water-supply. For thirty-four years the institution had used its own spring ; but at the time of the outbreaks recourse had been had to river-water in addition, pumped up directly from the river into the

¹ ‘Fluid in the pericardium is diagnostic of beri-beri’ (Pekelharing and Winkler). ‘The pericardium nearly always contains an excess of fluid’ (Manson, Davidson, p. 475).

hospital tank. A chart given shows that on each of the three several occasions when this supplementary supply was drawn upon there was an outbreak of the disease coinciding with its use both in time and very closely in extent with the amount of water taken from the stream. If this water were really the source of the neuritis, it is clear that the neuritis was not beri-beri, for in the case of the latter many experiments have finally eliminated water from among the factors possible in its etiology.

Arkansas State Insane Hospital Epidemy.—Bondurant quotes a description supplied him by Dr. W. B. Barner, Assistant Physician, of a small epidemy of cases similar to the Tuscaloosa ones, which happened in July, August, and September, of 1895, at the Arkansas State Asylum, Little Rock. 'The cases numbered between twenty and thirty. . . . The prominent symptoms were malaise, with rise of temperature to 100° to 102° F in the afternoon (a degree less in the morning), œdema of *ankles*, loss of patellar reflex, and foot-drop. In some cases the limbs were hyperæsthetic, in others partial anæsthesia was observed; some suffered much pain, others none. The gravity of the cases varied, the milder showing œdema of ankles with ill-defined paralytic symptoms, the severe ones complete motor paralysis of the legs. Only one case proved fatal. . . .'

Bondurant again claims these cases as beri-beri, though it would seem that Barner had not done so. Again, it must be repeated, if it were beri-beri, the type was modified; and, again, here also there is no exclusion of the possible effect of rice.

In this case also there was likelihood that bad water was the source of the malady.

At Saint Gemmes-sur-Loire there occurred in 1898 an epidemy among inmates of an asylum, in which the earlier signs included an erythematous eruption on the face and hands, with trophic sores, bladder and rectal palsy, and general rapid ascending flaccid paralysis. The reporters, Chantemesse and Ramond,¹ designated this malady as 'recalling' (*rappelant*) beri-beri. Norman has swept these epidemies in with others not like to the renal disease, to support the view that there was a widespread wave of infection about the time of the Richmond Asylum epidemy affecting many asylums besides his own. Thus:

At the Suffolk County Asylum at Melton, he states, there

¹ *Ann. de l'Inst. Past.*, September, 1898.

was such an epidemic in 1894-1895, and again in 1896-1897, of which, however, I have been able to obtain no account. Two sporadic cases of multiple neuritis were described by Orthmann as having occurred at the Grafenburg Asylum in 1897, and at the meeting where these were discussed Toppel mentioned others under his care in the same year at the Altscherbitz Asylum (near Leipzig). But I find in these accounts nothing to supply a reason for regarding them as beri-beri, nor did their reporters regard them as such.

I find that Scheube has expressed a similar view, suggesting the affinity of these and many sporadic cases of polyneuritis rather to the group of maladies of which anterior poliomyelitis is the commoner expression, as in the epidemics related by Eisenlohr in Hamburg and Protopopow in Russia. Bondurant significantly makes reference to the same relationship, and mentions an epidemic of such cases happening in Rutland County, Vermont, in June and August, 1894. These cases were investigated by Dr. C. S. Caverley. There were 'upwards of 130 cases,' almost all occurring in one valley, at the end of a very dry season, the water-supply, direct from the stream, then very low, being sewage-contaminated, as it was in the other epidemics he details. In this epidemic 'most of the cases occurred in children. . . . Many domestic animals—horses, dogs, fowls—were also affected.'

'The chief symptoms were motor paralysis in arms or legs, with minor sensory disorders, and much cerebral disturbance, leading in some cases to the diagnosis meningitis,' so that there was less than the usual resemblance to beri-beri. But the reflection is provoked that in the case of so-called beri-beri arising in asylums, the presence of cerebral signs, the absence of which is so characteristic of the true disease, would be likely to be assigned not to the intercurrent but to the prior mental disorder; thus a point of distinction would be apt to become overlooked.

Yet of the same epidemic Dr. Caverley wrote to Bondurant: 'There is no doubt some of the cases in our epidemic of 1894 were neuritis.'

'A similar outbreak, on a smaller scale, occurred,' Bondurant further states, 'during the summer of 1896, in Greene County, Alabama, about forty miles south of Tuscaloosa.' These cases were 'fifteen or more in number, scattered over an area of country ten to fifteen miles in diameter. Adults as well as

children were attacked. The disease began with fever and systemic disorder. . . . A few days to a week or two after the fever appeared some paralysis developed, involving sometimes one upper or one lower extremity, sometimes both extremities on one side, sometimes both upper or lower extremities, and in one or two instances both lower and one upper extremity. Most of the patients recovered *entirely*—not a common feature in poliomyelitis—and all improved, but in some there still is, seven to nine months after attack, some paralysis and muscular atrophy remaining. . . .’

‘Two or three cases of infantile paralysis have occurred near Tuskaloosa during the past twelve months, but there has been nothing approaching an epidemic of the disease.’

Though Bondurant would not include these as cases of beri-beri, it appears to the writer that they resembled the malady in the Bryce Asylum, at least as closely as the cases there did beri-beri; and that all the evidence he has presented only goes to show that there was at that period a widespread epidemic of a disorder more nearly allied to poliomyelitis than anything else, and that this was the disorder which prevailed at the asylum.

In none of these epidemics or sporadic cases, it may be worthy of note, was *rice* excluded as a possible factor; it was not considered.

The outbreak next to be referred to is interesting as the latest epidemic occurring on land in a temperate climate which has been claimed to be beri-beri, and is one in the causation of which it is also asserted that food, including rice, could have had no share.

Epidemy among Boer Prisoners at St. Helena.—The epidemic at St. Helena occurred during 1901-1902, and was described as follows by Casey:¹

‘On taking up duty at Deadwood Camp, St. Helena, on March 1, 1902, I found quite 80 per cent. of the Boer prisoners applying for treatment suffering from a pronounced œdema of the lower limbs. In some cases the entire body, including penis and scrotum, was œdematous. There was generally great debility, marked pallor, and weak and excited cardiac action. The urine was free from albumen. There was wrist drop and foot drop, with absence of patellar reflex, in most cases. Those patients

¹ *An Epidemic of Beri-beri in St. Helena*, by J. P. Nunan Casey, L.R.C.P. and S. (Edin.), L.A.H. (Dublin), late Civil Surgeon, Imperial Forces.

who appeared to be in a later stage showed greatly impaired sensation and power of motion in legs, and complained of cramp in the calf muscles, as well as of pain and sensation of weight in chest. In a few cases sensation in the legs was totally lost, and in most greatly delayed, and this was found also in "dry" cases with little or no œdema, the main commencing complaint of the patients being that they could not feel properly in their legs, or that their legs were sore. Deep pressure on the calf muscles always revealed tenderness.

'I diagnosed these cases as beri-beri, from the similarity they presented to those of that disease that I had previously seen in South America, more especially in North Brazil and the Amazon region. Although most of my colleagues differed from the diagnosis, it was confirmed by Colonel Williamson, P.M.O., when he paid his official visit to St. Helena. He pronounced the cases genuine beri-beri. It may be mentioned, *en passant*, that the circumstances of the sufferers being Boer prisoners put the question of alcoholic neuritis out of the question.

'I treated them with a single dose of calomel, followed by a mixture consisting of Mag. Sulph., Pot. Acetat., Sodæ Salicylat., Tr. Digitalis, Tr. Nux Vom., Chloric Æther, and water, given three times a day. To this I added later, on the suggestion of Colonel Morse, S.M.O. at St. Helena, Nitro-Glycerine solution in 3-minim doses. A liberal diet was allowed, with lime-juice, and 2 or 3 ounces of brandy per diem.

'Under this treatment and dietary the patients usually improved, and more markedly when they were transferred to fresh ground away from other prisoners. Lead lotion locally seemed to relieve the *burning in the feet*. Some few cases grew steadily worse and died, some rather suddenly. One case was complicated with pneumonia. From information gleaned on the island, I have reason to suppose that beri-beri was formerly more at less endemic there, which renders it possible that some local toxic nidus might still be operative. Overcrowding, monotony, and insufficient diet, may have been contributory causes. I had never any doubt about the diagnosis.'¹

Mosse² has also more recently given an account of the same

¹ Extracted from the *South African Medical Record*, vol. i., No. 7, p. 107. I am indebted for this account (copied from the office file) to the courtesy of the manager, C. L. Darley Hartley, Esq.

² *Journal of the Royal Army Medical Corps*, September, 1904.

epidemy from which the following additional information is, gained :

There were 3,500 prisoners, among whom were 91 cases with 5 deaths.

Mosse notes that 'signs and symptoms of a peripheral neuritis were usually present in a greater or less degree. Patients complained chiefly of burning in the feet and legs, and numbness. . . . In paralytic cases the ataxic gait was well marked.' There was in some 'loss of power, with inco-ordination of movement.' The dry form, and cases with enormous anasarca, are illustrated by photographs. There was variable anæsthesia, and no cranial implication. 'The digestive tract was not usually affected, appetite was good, and the tongue clean.' Vomiting was noted in two cases. The urinary system was almost invariably normal. Among points of 'special clinical interest' to which Mosse draws attention, he mentions the sudden appearance and disappearance of anasarca, and the 'rapid recovery of the purely paralytic cases with ataxic symptoms.'

Can it be said that the picture composed by these two observers is one of typical beri-beri? Can it be said that the disease described here is the same as that at Dublin or Tuskaloosa? The resemblances—anæsthesia, paræsthesiæ, paresis, atrophy—which were noted in the St. Helena epidemic are, again, those common to all peripheral neuritis; but it is clear that the paresis was neither frequent nor extensive. It certainly could not have amounted to bad paraplegia, or tetraplegia, or it would certainly have been mentioned. There is nothing as to the flaccidity of limbs, the relaxation of joints (so marked at Dublin), of the *pirri-pirri*, and the *shiyo-shin*, although the disease was acute enough to prove fatal to five of the ninety-one attacked. 'Burning feet,' it may be remarked, so conspicuous in ergotism, acrodynia, erythromelalgia, and allied disorders, is also a conspicuous and almost constant feature of Macleod's disease—epidemic dropsy—which in some respects these cases at St. Helena resemble, and which has certainly prevailed in Africa and many Pacific islands, and at Ascension in particular.

Moreover, rapid recovery from any, except the slightest, attacks is certainly not a common feature of beri-beri, while normal urinary function is almost unknown.

Lieutenant-Colonel Mosse has very kindly given me the diet of these prisoners, which was exactly the same as for the soldiers who guarded them.

DIET SCALE, PRISONERS OF WAR AT ST. HELENA.¹

Bread	1½ lb.	} Daily; but every Monday and Friday, in lieu of bread and fresh meat, 1 lb. of biscuit and ¼ lb. preserved meat.
Meat	1 lb.	
Coffee	½ oz.	
Sugar	2 oz.	
Salt	½ oz.	
Pepper	¼ oz.	
Milk (condensed)...	1 tin.	} On alternate days
Potatoes	½ lb.	
		or			} or
Mixed preserved vegetables	1 oz.	
					Every Monday and Friday.

Commenting on the etiology, Mosse states that both soldiers and prisoners had exactly the same rations, and he holds this fact to exclude the possibility of the disease having been in any way dependent on the food. He considers that the greater dirtiness of the prisoners, their being overcrowded, and the fact that they were many of them profoundly depressed, were pre-disposing causes which determined their infection by a germ, the growth of which he supposes was also favoured better in their camp than in the soldiers' quarters.

To this, of course, it may be objected that the depressing influences able to determine the growth of a germ in an otherwise resistant person may equally well be supposed able to pre-dispose him to succumb to the action of any poison, whether derived from food or otherwise. Moreover, one of the points upon which Mosse specially lays stress is against any germ action. He says: 'It was remarkable the improvement which immediately followed the removal of those affected to a more open camp on new ground.' This is an old point often discussed. How could such a change so affect the action of a germ already within the system? But with this change of place came at the same time improvement of diet. 'As soon as the disease was recognised . . . a generous dietary was allowed, the chief extras given being oatmeal, lime-juice, milk, and alcohol.'

While the records leave it doubtful whether or not these cases of peripheral neuritis at St. Helena were modified beri-beri, they afford no ground, in the writer's opinion, for Mosse's assertion that food was excluded from the causation.

These three epidemics, none of which can be asserted with certainty to have been beri-beri, are the only ones the writer has been able to find recorded occurring on land in the causation of

¹ This diet may also be said to have been (the upholders of Brémauds' theory will certainly point out) lacking in fat, unless the meat supplied had plenty.

which it seems that rice directly consumed as such for food may have had no share.¹

It is obvious, seeing that the greatest extremes of climate, considered as factors *per se*, have been proved to be no bar to the development of beri-beri in typical unquestioned form, and seeing that the field for its activity—all the populations of the world outside the tropic zone—is extensive, that the sparseness of the recorded outbreaks ascribable to the beri-beric noxa makes the case for beri-beri of any other than a rice origin very weak indeed. For there is hardly any factor of hygiene, apart from the climatic ones, not shared alike by all the various races of the world. Dirt, overcrowding, and underfeeding prevail in all parts alike. Be it a question of importable infection, then the incessant world-traffic of the day permits the spread of every infection, as witness cholera and plague. Scheube² has even ventured to postulate, as a source for the Richmond Asylum epidemic, the introduction of contagion through Dublin as a port! Yet beri-beri cases are landed continually in nearly every harbour of importance without any such sequel, and in any case the infection view has (it will probably be conceded) been finally disposed of in this work. How, then, explain the complete absence, through all historical times, of so easily carried a malady as beri-beri from all the temperate regions? There is only the food factor, the national difference in food habits, which will explain this. It is only because they have not been rice-eaters that the folk of the temperate latitudes have remained, and by virtue of such abstinence that they are alone likely to remain, free from beri-beri.

I have just referred to a complete exemption, ignoring the apparent exceptions just discussed. But if these epidemics be claimed, be ever proved to be beri-beri, even in modified form,

¹ Ninety-one individuals among 3,500 of the prisoners (2·6 per cent.) got beri-beri. Mosse assumes it to be certain that they got no rice—ate, in fact, exactly the rations which were supplied them. But, if one reflect on the facts—(1) That the prisoners were of a class heavily addicted to smoking, and often to drinking also; (2) that they got no allowance whatever, either of tobacco or spirits, or cash wherewith to buy these things; (3) that the ordinary principal food of the native residents of St. Helena is rice; (4) that among the natives meat is scarce; it becomes an obvious suggestion that the few Boer prisoners—two to three in every 100—who did get beri-beri, may have given or sold their meat or other Government rations to the natives, taking the cheaper rice and cash, spirits, or tobacco in exchange. Could Lieutenant-Colonel Mosse be certain that this did not occur?

² *Loc. cit.*, p. 263.

they may yet be merely the exceptions which will prove the rule. It has been asserted, but in nowise proved, that rice played no part in these epidemics.

But there need be no precipitancy in assuming that epidemics in which rice is not *directly* traced are therefore wholly unconnected with rice in any form ; nor, if all possible derivation from rice be disproved, and the source of the disease in such cases can be traced to no other grain, is it even then necessary to assume the disease not to be due to grain-intoxication, nor to be beri-beri.

In the case of *pellagra* (due to maize-eating) neither the chemical poison, which produces the symptoms, nor the vital agent, which produces the poison, has yet been identified, and yet the correctness of the ascription of the disease to *something usually found in maize* is not doubted ; nor, if cases of *pellagra* occur, in which no connection with maize can be traced, is it to be doubted that they are the result of the same agent, though operating through other media.

So, in the case of beri-beri, neither the poison nor its producer may be ever identified in rice, and yet rice may be accepted as the usual cause, though not necessarily the sole one ; and cases in which rice is not concerned must be accepted as due to the same agent acting through other channels.

Before considering what substances, other than rice, may carry the beri-beri poison, it may be as well to consider what ways there are, other than consumption in its ordinary recognised form, by which rice itself may become operative in epidemics with which it *apparently* has no connection.

Indirect Modes of Rice Consumption—Adulteration.—Although it is certainly in rice consumed as such for food that the toxic agent is usually ingested, it by no means follows that this is the only mode in which the same thing can be presented or absorbed. There are other less obvious ways, other materials, in which rice or the poison causing beri-beri can be, and probably are often, unconsciously consumed. In some of these *rice* itself is presented under another name, or appears as an *adulterant* ; in others what may be termed *extracts*, or *derivatives* of it, are concerned.

Wheaten flour is frequently adulterated with rice. The commodities known as ship's biscuits do not bear too close a resemblance to those known to be made from wheat. Vorderman¹

¹ 'Onderzoek.'

states he has seen excellent 'bread' and ship's biscuit made of a mixture containing one-third by weight of rice offal.

'There is an article,' Hassall¹ says, 'in common and daily use by bakers, denominated "cones," or "cones flour."' Genuine 'cones flour,' he explains, is supposed to be made from a particularly fine quality of wheat called *revet*. It is used to dust over the surface of dough when kneaded, and to face the sponge before baking. A sample of this stuff seized at Peterborough, and sent Hassall for analysis, proved to be *entirely rice-flour*. He then procured more samples, and of twenty-two, only five were composed of wheat. The rest were made up of various adulterants, such as rye, rice, beans, maize, etc., 'the rice being the most frequent constituent.'

'*There can be no question,*' Hassall states, '*that cones flour is frequently employed in the adulteration of bread.*'

Heret² states that 'flour is continually adulterated with potato, rice, maize, oats, barley, or rye,' but that 'rice is only added to wheat-flour exceptionally (in France).'

Winter Blyth³ says: 'In England adulterations of flour are of extreme rarity, with perhaps the exception of potato-flour and *ground rice*.'

Rice is also added, boiled, to the dough used for bread, according to Hassall and other authorities (Wilson, Thompson, Stevenson and Murphy).

Being able to hold about 77 per cent. of its own weight of water, and to retain much of this in baking, more tenacity is given to the dough by the addition of rice; its 'rising' properties are increased, and a much heavier loaf produced with less flour.

To what extent adulteration of bread and flour with rice is actually practised by bakers or millers is, naturally, not easily ascertained.

The retail price of rice suitable for the table in England is slightly cheaper than that of good wheat-flour. A *good sample* of the former may be got at 9s. 6d. per hundredweight (1.01d. per pound), as compared with wheat-flour at 22s. per sack of 252 pounds (9s. 8d. per hundredweight, or 1.05d. per pound), an example taken from a tender for a workhouse supply. The wheat-flour suitable for making good white bread is seldom priced below 13s. 9d. per 140 pounds (1.2d. per pound); while rice *suitable for*

¹ 'Food and its Adulterations,' p. 317.

² 'Dict. des Altérations et Falsifications des Subst. Alimentaires,' Paris, 1895.

³ 'The Composition and Analysis of Food,' 1903, p. 153.

adulteration can be obtained greatly cheaper than this—6s. or 7s. per 140 pounds, or $\frac{1}{2}$ d. per pound. It is obvious that there are both facilities and temptation to adulterate. Dr. Hassall also states that some of the best *arrowroots* are made entirely of rice-flour. He mentions that Messrs. J. and J. Colman largely manufacture such an article.

These unrecognised modes of rice-consumption are highroads by which beri-beri may be introduced into regions other than the tropics ; and there are by-ways.

Glucose.—There is a product, largely derived from rice, which is widely consumed in unsuspected forms, being used as a substitute for cane-sugar in the preparation of many sweetstuffs, and also in the making of what is now called 'beer.' This is 'starch-sugar'—dextrose or glucose.

In England enormous quantities of this stuff are used annually. In 1902 1,147,520 hundredweights of it were *imported*. In America, the chief source of the imported supply, commercial glucose is made from corn (maize). In Germany and Belgium, from potatoes. But in England, O'Sullivan says, 'the materials chiefly employed in the manufacture of glucose are sago, maize, and *rice-starch* ; *finely-ground rice* itself, as well as granulated maize and rice, being at times used.' The cheapest and usual source is rice.

What the amount of glucose made in England from rice or other material may be I can find no figures to show ; but the total quantity of rice imported into the United Kingdom in 1902, very little of which was exported again, was 5,398,310 hundredweights. Much of this (1,579,477 hundredweights) was described as 'other than whole and cleaned.'

The consumption of rice as an article of diet in England, though growing, is yet not large. The chief commercial use of the grain is to make sizing for cotton goods, starch for all purposes, and British gum (or dextrin). But much of it goes to the making of glucose.

The process of glucose manufacture depends on the fact that starch when treated with dilute acids is hydrated and dissolves, dextrin and maltose being produced ; then, by the further action of acid, dextrose.

One of the methods in use commercially consists in boiling the material with sulphuric acid and water (in the proportion of 100 of solid, 250 water, 5 acid). In an hour and a half to two hours conversion of the starch to dextrose is complete.

In another (Johnson's) process the raw rice is first allowed to saturate itself with water containing 2 per cent. of hydrochloric acid, washed, steamed at a pressure of 75 pounds, diluted with an equal volume of water at 150° C. (injected into the special receptacle under pressure), and the conversion is effected in one and a half minutes. In a third (Soxhlet's) process residues from the manufacture of starch are treated first by boiling till gelatinized, then by cooling, adding a malt-infusion, reboiling, and treating with dilute acid.

The liquor resulting from the different processes is neutralized with chalk, filtered through bags, etc., decolorized with animal charcoal, concentrated in vacuum pans, and crystallized in iron moulds.

Commercial glucose so made is of variable, dirty yellowish colour, and usually contains, in addition to the dextrose, small quantities of maltose, dextrin, unfermentable organic bodies, and ash. 'The proportion of unfermentable bodies is sometimes as high as 15 per cent.'¹

I have referred thus at length to the methods by which glucose is prepared from rice, that it may appear how easily they may lend themselves to the distribution of any poison which might be found in rice, provided it were non-fermentable, and not volatile. For the methods followed in making of glucose are just such as serve, in dealing with other material, for the extraction from them of their various specific properties.²

Supposing the poison, to the presence of which in rice beri-beri has been shown to be due, to be a body resembling ergot or an alkaloid, is it not likely that it will be extracted from rice containing it in the preparation of glucose, appearing perhaps in the 15 per cent. of unfermentable extracts which O'Sullivan has stated to be commonly present?

Distributed and consumed through this channel, in instances favouring the continued use of contaminated articles, its usual effect will ensue, and epidemics or sporadic cases of beri-beri will be produced, which are none the less due to rice, although the agency is indirect and the derivation remote.

The applications of glucose are various. Its use as a substitute for cane-sugar in such messes as modern jams, 'golden

¹ Corn. O'Sullivan, F.R.S., etc. Thorpe's 'Dict. App. Chem.' article on Glucose.

² Quinine is thus extracted from bark, strychnine from nux vomica, morphine from opium, etc.

syrup,' etc., is probably more extensive than is suspected. The principal purpose for which it is used is as a 'brewing sugar'—one of the many substances for malt in making beer.¹

Dr. J. Hereon² says: 'Rice and maize are used as adjuncts to malt in brewing' and as 'substitutes for malt.' Rice is used either raw, as gelatinized rice, or as glucose derived from rice.

Forbidden in Germany, but permitted in England, the use of these adulterants and substitutes for malt—euphemistically termed 'adjuncts'—has become so extensive of late years, that from forming only 6 per cent. of the material used for brewing in 1878, in 1900 they formed 22½ per cent. The actual quantities were:

			Malt.			Sugar, Rice, and Maize Substitutes.		
			(Bushels.)			(Bushels.)		Per Cent.
1878	59,388,905	3,825,148	...	6.05
1900	57,354,904	16,727,536	...	22.56

It is stated that in most cases the sugar is used for what is called 'priming'—*i.e.*, adding body and brilliancy to the beer.

In this case impurities in the glucose would inevitably contaminate the beer, the stuff being simply dissolved in it. But even when the glucose, used purely as a substitute for malt, is all fermented, it is clear that anything present in it which is not fermentable, and which is at the same time soluble (to even a small extent) in water, will remain in the beer.

Quilter,³ in the House of Commons, stated that the amount of *sugar*⁴ alone used in brewing, under 2,000,000 pounds in 1856, had risen by 1894 to over 245,000,000. Two firms, who brewed 800,000 gallons between them annually, used in 1882 576 bushels of malt to 1 of sugar; but in 1894 only 358 bushels to 1 of sugar.

Of 130 brewing sugars analyzed by the Royal Commission on 'Arsenic Poisoning⁵ in Beer Drinkers,' 63 were glucose.

Of 37 samples of 'malt adjuncts,' 10 consisted of rice.

Of the quantities of glucose used the following table, calculated from figures given by the same Commission, will afford some illustration. It shows the proportions of glucose used in brewing

¹ 'Encyclopædia Britannica,' tenth edition, article on 'Brewing.'

² Thorpe's 'Dict. App. Chem.,' vol. i., article on 'Brewing.'

³ Reference: *Lancet*, May 18, 1895, vol. i., p. 1284.

⁴ From all sources, not rice only.

⁵ Report, 1903.

the quantities of beer stated at several breweries, samples from which were taken by the Revenue officers.

Pounds of Glucose used.			Gallons of Beer brewed with it.			Grains of Glucose used for each Gallon of Beer produced.
336	1,703	1,381
672	4,904	904
1,120	7,223	1,085
672	5,898	797
224	5,961	262
224	1,466	1,069

Hammond Smith,¹ in evidence before the same Commission, stated :

‘Glucose is an important ingredient of numerous articles of food, particularly table syrups, marmalade, confectionery, and biscuits, also of sundry beverages, particularly brewed ginger-beer and certain kinds of wine.

‘In most of these foods and drinks the proportion of glucose used may be considerable.

‘Nearly all the glucose used in these substances is of foreign, usually American, manufacture.’

Referring to ‘golden syrups’ and treacles, he says :

‘The quantities of glucose which are occasionally employed are illustrated by certain recent prosecutions, in which a mainly glucose syrup was sold as golden syrup or treacle. For instance, on December 19, 1901, a grocer was summoned at Southwark for selling as golden syrup a substitute containing 15 per cent. cane-sugar, and 85 *per cent.* starch glucose.’

‘At Fenton, on April 30, 1902 (*Food Journal*, May, 1902, p. 117), a retailer was summoned for selling as treacle a substance which contained 70 *per cent.* of glucose.’

In jams and marmalades ‘such amounts as 5 to 10 per cent. are common.’

Glucose is used in sweets. ‘Some, such as chocolate and other creams, consist largely of glucose.’ Tested samples showed proportions of it as much as 20 to 33 per cent.

Glucose, Smith also informed the Commission, is ‘a frequent ingredient in the manufacture of what is termed “hard dough” for making a certain class of biscuits. . . . One of the objects is to impart a fresh-looking appearance to the biscuit. The proportion of glucose used was given me as 6 pounds of glucose to one sack of flour, producing 360 pounds of biscuit’ (1.7 per cent.).

‘Malt extracts and yeast foods are ordinarily employed in bread-making to the extent of 1 pound of extract to 28 pounds of flour.

¹ *Loc. cit.*

'Several so-called "malt extracts" consisted partly of glucose.

'One such, I am informed, consists of glucose, pea-flour, cane-sugar, and phosphates.'

All these are so many channels through which rice-derived poison, causing beri-beri, may be distributed widely in unsuspected guise. In bread and biscuits made from rice-adulterated flour; in modern English 'beer'; in jams, syrups, etc., it is possible, and even likely, that the source lies of many of those epidemics on ships and in asylums in temperate latitudes which have hitherto seemed to oppose an insuperable obstacle to the acceptance of the rice-theory of beri-beri.

Such are some of the indirect ways in which rice, or poisons derived from rice, may become able to produce beri-beri. It is obvious that they may operate equally in temperate latitudes, at ocean, or in the tropics.

In none of the records of epidemics, whether in Europe or America, or at sea, in which rice has been declared to play no part, is there any evidence that any of these avenues for its action has ever been examined.

Hence, the assertions of Ucherman and others that *beri-beri occurs independently of rice*, so far as they are based merely on the observation that cases occur where rice has not been consumed *in the ordinary manner*, are not justified by the facts, and must be held to be unproved so long as the various modes of access of rice which have been indicated remain unexplored.

But accepting this entirely unfounded statement as representing facts, assuming that cases of beri-beri, or epidemics of it, may happen, with which *rice cannot* in any way be concerned, they are still simply enough explained as instances of *grain-intoxication*.

Rice Toxin in other Cereals.—When, following plain logical method, we seek for the common factor which must exist between the isolated outbreaks of beri-beri in temperate lands, the cases on ships, and the thousand daily epidemics of it in the tropics, it is easily seen what that condition (necessarily one of *food*) must be.

Other than cereal grains, there is no sort or class of food-stuff eaten by the European which is ordinarily represented in the dietary of the tropical native. Certainly the only constant factor, the unique condition common to absolutely *all* the epidemics, all the cases (of which the facts have been recorded) has been the use of cereal grain.

It is unnecessary to recapitulate here the reasons (already

adduced) which make it probable that a parasite, a poison, affecting one species of a genus of plants should at times also attack other members of the tribe.

The fact that a poison has been conclusively shown to be developed in one of these—rice—and to be the cause of beri-beri to thousands of its users, leaves little room for doubt that other grains are subject to the same change, the development of the same poison. When to this *probability* is added the *fact* that all that is common between beri-berics in all regions is the consumption of grain, the conclusion that the disease (beri-beri) is due to the same poison operating through the different grains eaten in each case is an induction from the facts so sound that little (short of demonstration of the actual toxic substance present in all the grains) can be added to it in the way of proof.

The consideration that in many of the cases of beri-beri not dependent on rice the articles of food, when examined, have appeared sound, so far from weakening this conclusion, on the contrary, supports it. Since, were the disease due to change of the grain (bread, biscuits, or flour of wheat, barley, oats, or rye) by common appreciable decay, epidemics of beri-beri in temperate latitudes must have been far more frequent, and the connection of them with damaged grain (which is common enough) must have been noticed, as has been the case with ergotism and pellagra. While, on the other hand, it has been shown that rice which has appeared to be dry and sound and sweet has undoubtedly contained a poison producing beri-beri.

As common decomposition of food-stuff is no actual *indication* of the presence of the specific poison of beri-beri, then, so neither does apparent soundness contra-indicate its presence. But the facts of decay being so far a sign of neglect, of age, of adulteration, or of inferior quality of the food-stuff, point to the greater chance there is that such articles may have become infested with the specific beri-beri poison also.

In considering the circumstances under which beri-beri chiefly occurs apart from rice—namely, upon ill-found ships and in poorly-dieted asylums—it will be seen that the conditions certainly favour the adulteration of the better cereals with cheaper rice, and the provision of cheap and therefore probably diseased grain instead of flour of good quality.

One of the deductions from the rice theory, as it was formulated earlier in these pages, was that, as must almost necessarily result from any form of poisoning which affects common staples

of food, 'it should be apt to affect most often and most extensively public institutions in which many persons are housed together.' The reason for this being, not factors of overcrowding or association, of restraint or of locality, but merely the economic one, that in such places the diet, necessarily of the cheapest, is most likely also to consist of inferior, and therefore often probably adulterated, articles.

In the tropics the special proclivity of all such institutions to the disease is illustrated by abundant instances, many of which have been mentioned. Elsewhere, and in temperate climates, it, or at least a malady closely simulating it, has affected, not indeed prisons, but asylums and ships—the conditions of life upon which perhaps combine the features of both.

In British Malaya the worst 'foci' of the disease, when its cause prevails, are the asylums at Singapore and Kuala Lumpur.

This peculiar severity of incidence of beri-beri on the *insane* seems to imply the existence of some factor not shared by inmates of other institutions, and calls for comment. Is the special liability due merely to the greater susceptibility, commonly observed, of lunatics to nearly all diseases? To some extent, no doubt, this is the case; but all that is thereby shown is their lessened resistance to the disease when introduced. The greater tendency of the malady to *appear* among them it is which most requires to be explained. A solution is suggested by an observation made by Norman, mentioned in his account of the great epidemic of beri-beri at Dublin, which he refers to as inexplicable, although upon the food-theory of the disease it is easily enough understood. It is that, as he himself observed at the Richmond Asylum, and as Ramond and Chantemesse also noted at St. Gemmes-sur-Loire, among inmates of asylums, the *epileptics* are much more prone than others to the disorder. Now, the peculiar sensitiveness of the epileptic to the influence of food, his ready reaction to slight differences in diet, as well as his frequent gluttony, are well known, and it is to be expected, therefore, that unusual poisonous constituents of diet should affect this class sooner and more severely than others.¹ In the Richmond Asylum there were some differences between the rations of the epileptics and the other inmates.

In the French asylum it was also noted that the poorer patients,

¹ Special diet is usually given to epileptics, in which there is less meat and relatively more carbohydrate. This would tend to increase their chance of being affected by poisons conveyed in the latter.

whose *rations* were inferior to those of the others, were more affected.

At the Tuscaloosa Asylum, Bondurant¹ states: 'It was, generally speaking, not the physically but the mentally enfeebled who fell victims to the disease. There were eighty epileptics in the hospital; of these, thirty-two had beri-beri. The remaining thirty-nine cases occurred in imbeciles, paranoiacs, and those terminal dementals showing marked degenerative stigmata. No patient having an acute or curable form of insanity took the disease; no one of the 600 or 700 patients actively employed in work on the farm, in shops, laundry, or elsewhere was attacked; and no case occurred among the 200 employees of the institution.'

In the case of this epidemic also, then, of modified beri-beri, if such it were, the strongest factor determining attack was a certain condition constituting predisposition to it in the most certain sense on the part of the individual.

These reflections tend to the suggestion that similar sensitiveness of reaction to slight differences in food may characterize the insane generally, as compared with healthy persons, and that in this susceptibility may be found an explanation of the special tendency of asylum inmates to get beri-beri.

It seems reasonable to suppose that those whose nervous systems already are, from whatever cause, unstable, should suffer in greater degree than others when exposed to an influence itself producing general nerve decay.

Thus the asylum populations would be likely to be first, and often solely, attacked in communities otherwise free, not because they alone were exposed to or consumed the toxic agent, but because they succumb to doses too small to affect the healthy, the poison being taken in equal degree by all.

There are other analogies supporting such a view. The greater tendency, for instance, of alcoholics than the temperate to almost every form of disease is an axiom with actuaries, while Oppenheim² has called attention to their greater liability, when employed as metal-workers, to the diseases associated with such trades. Raw³ cites Osler as having stated that arsenic more frequently entails injurious effects when given in alcoholic solution, and Reynolds has insisted that the operation of each agent is more virulent when associated with the other.

When to what may be called such a 'specific tendency' of the

¹ *Loc. cit.*, p. 685.

² *Loc. cit.*

³ *British Medical Journal*, October 12, 1901, p. 1044.

insane to succumb to other neuropathic agents are added the influences of inferior, if not actually insufficient, feeding, and the depravation of habit which attends overcrowding, the concurrence offers a minimum of resistance to the action of the toxic agent which, once introduced, may be expected, even in small doses, to produce disastrous effects.

Ship Beri-beri.—But if the subjects most susceptible of beri-beri, when its cause is abroad, are the inmates of asylums, it is on shipboard that the agent of the disease is likely to be applied oftenest, and with great effect. For though the sailor, exposed usually to harder toil and influences more depressing than those attending almost any other calling—extremes of heat and cold, frequent and prolonged wet and chill, close confinement, overcrowding, monotony, lack of regular exercise—naturally requires better feeding, it is a discreditable fact that the food supplied to him is commonly insufficient, often unwholesome, and sometimes even decomposed. In the lowered state of the system brought about by prolonged courses of such improper food, when the specific toxic agent of beri-beri is presented, little resistance is offered to its action, and the subjects readily succumb.

It is probable, too, that on ships 'found' at the cheapest possible rate, old, ullaged, and adulterated articles of food of all kinds find place.

The majority of recorded epidemics on British ships occur among Lascar crews, whose rations consist principally of rice, the allowance of which (24 ounces) is, in the writer's opinion, excessive absolutely, and relatively greatly so, forming 53 per cent. of the total dietary at sea, and 43 per cent. of it when in harbour.¹

¹ The regulation diet is as follows :

SCALE OF PROVISIONS FOR LASCARS OR OTHER NATIVE SEAMEN
(BOARD OF TRADE REGULATION).

	Pound.	Ounces.	Drachms.	
Rice	1	8	0	= 24 ounces.
Dhal	0	4	0	} = 31 ounces.
Ghee	0	2	0	
Salt	0	0	8	
Salt fish (at sea)	0	4	0	
Curry stuff	0	0	10	
Fresh meat (in harbour)	0	10	0	
Vegetables	0	8	0	
Tea	0	0	4	} = 31 ounces.
Sugar	0	1	8	
Lime-juice (after ten days out of port)	0	1	0	

The proportion of rice in this dietary would be 32 per cent. when in harbour, 44 per cent. when at sea.

One may infer from figures supplied by Rees¹ (already quoted) that on at least half of the ships upon which Lascars are employed there is almost constantly beri-beri. In view of this diet it is rather a matter of surprise that it is not so on all.

Japan has pointed out the remedy in this matter. Saneyoshi² states that in the N.Y.K. Fleet beri-beri disappeared on attention being paid to the diet of the sailors.

The derivation of beri-beri by the Lascars from *rice* is obvious, but it is otherwise with the many, and increasingly frequent, epidemics on ships manned and found entirely by Europeans.

Is the Beri-beri of Ships True Beri-beri?—As has been made sufficiently apparent by the data already submitted, it is more than questionable whether any of the epidemics of so-called beri-beri observed on land in extratropical zones can properly be included with the tropical disorder. At the most some of them may be regarded as modified beri-beri, evidence of the operation possibly of the same agent as causes the true malady, under circumstances modifying its action, or of the operation of a closely similar but really distinct poison.

But this being admitted as regards the epidemics on land, it is obvious that the epidemics aboard ship are no less likely to belong, some of them, to the same or similar categories.

The writer has, it will be noted, hitherto throughout this work appeared to accept, or at least have discussed without entering any caveat to the contrary, all the ship epidemics called beri-beri as the real disease.

This course was chosen since the cases used for illustration, as it seemed, actually were so, being instances of outbreaks dependent on rice, and so conformed with the theory, while it could not have assisted but would have cumbered the main argument if, at an earlier stage, the diagnosis of ship beri-beri had been generally challenged.

But both from the consideration stated above—the analogy of the land epidemics—and from a careful perusal of the very numerous accounts of the ship cases which are available, the critical must be led to the conviction that the same fallacies which have obscured the issue on land have been equally, or more so, sources of error at sea; that a large (if not the greater) proportion of the cases of disease on ships which have been designated as beri-beri certainly do not conform to the type, and must be

¹ *British Medical Journal*, 1897, vol. ii., p. 747.

² *Loc. cit.*

regarded as not even a modified form of the disease, but as a distinct if closely allied disorder.

Upon ships, as elsewhere, true beri-beri may, and doubtless often does, occur. But the commoner malady, that for which it is frequently mistaken, bears to it, there is ample evidence to show, only a superficial resemblance, and is distinguishable by marked and almost constant features. At the same time it is, naturally, also liable to be *complicated* by the real disorder.

In both diseases dropsy is a common factor ; in both signs of neuritis also occur. But while in true beri-beri the former is rare, and the latter predominates, in the ship disease the reverse conditions prevail. And this difference obtains not merely occasionally, but, viewing the cases of either sort in the groups in which they occur as epidemics, it is a constant difference. Can two disorders distinguishable by a divergence so marked be classed as identical ?

Here a word of warning is necessary to prevent possible misconception.

It is not intended now to revive the old dispute, so long an obstacle to the identification of beri-beri as seen in different countries, whether the 'wet' and the 'dry' cases are manifestations of one and the same morbid agent. The established conclusion that they are so has already been recognised early in this work.

In true beri-beri, and except as a sequel, the result of failing circulation, œdema of any kind, certainly extensive dropsy, never occur *unaccompanied by other signs of nerve-implication*. And these changes, when the attack (as gauged by the dropsy) is severe, can be perceived not only by the physician—they speedily induce troubles which the patient also recognises, and of which he complains. But cases happen in which the presence of such dropsy early and in marked degree so overshadows and masks the onset, and later the development of the other symptoms—sometimes death intervenes—that other symptoms proper to the disease—its effects on the motor nerves—fail to be observed.

Although I have thus attempted to suggest a reason why the grave nerve-disturbance which underlies the dropsy in these 'wet' cases of beri-beri fails to be perceived, yet to the careful observer various significant signs will always indicate its presence. The voice, high-pitched, hoarse, whispering, or aphonic ; the facial anxiety ; the respiratory embarrassment ; the log-like limbs ; the wrist-drop of the raised arm ; the flaccid extension of the lower

members—all these make a picture which speaks of general nerve-paresis, unmistakably beri-beric.

The old problem was to explain the occurrence—not, after all, *common* in ordinary epidemics—of cases such as this, in which dropsy seemed to be the only or main symptom of a disorder the proper effects of which were even then recognised as being usually those of disordered sensation, of paralysis, and of atrophy.

The present difficulty is, on the contrary, to explain the *absence*, almost invariably, from epidemics of a disease reputed to be beri-beri of those very cases of paralysis, especially of paralysis with atrophy—‘dry’ beri-beri—which are its recognised, usual, and characteristic product.

Though late in the whole discussion, it will conduce to clearness in deciding the issue, which is very important, if we recall the definition of the disease, a sufficiently wide one, which we quoted from Manson at an earlier page (p. 5). Beri-beri is, according to this author, ‘*a form of peripheral neuritis, which occurs endemically and epidemically, and is especially characterized by . . . proneness to œdema and to implication of the neuro-muscular system of the central organ of circulation; by complete absence of trophic skin-lesions, of paresis of the muscles of the head and neck, of marked implication of the organs of sight, hearing, taste, and smell, and of the mental faculties; . . . there are troubles of locomotion, paræsthesiæ of various descriptions, especially in the lower extremities; marked hyperæsthesia of the muscles involved, with subsequent atrophy,*’ etc.

The signs of neuritis are the central phenomena, among which œdema is but one. The *absence* of certain lesions is significant. In another place Manson says of the œdema that ‘it begins *always* under the skin over the crest of the tibia’—an observation which many authors confirm (e.g., Pekelharing), but which the present writer holds to be true only so far as distribution on the limbs is concerned; for certainly in many cases the seat of first effusion is the genitalia.

Manson’s definition does not include the order of development of the varying symptoms, but in diagnostication of beri-beri recognition of the various signs when produced is not enough; the order in which they appear is even more important, since it is characteristic.

Pekelharing and Winkler¹ thus describe the symptoms recog-

¹ Bentley’s translation, p. 15.

nised at Atjeh by the experienced local surgeons as constituting the *initial phase* of beri-beri :

'Slight œdema along the crest of the tibia ; a puffy, pasty face ; difficulty in certain movements, observable only at first when the patient walks quickly or endeavours to go upstairs ; some paræsthesia or anæsthesia of the lower extremities ; palpitation of the heart ; a slightly quickened pulse, or, rather, a pulse which remains within normal limits while the patient is at rest, but which upon the least exertion goes up to ninety or a hundred beats per minute ; a marked contrast between the violent beats of the heart and the small and feeble pulse ; a prolonged first and reduplicated second sound of the heart. These symptoms form a complete summary of knowledge of the symptoms of beri-beri in the early stage.'

To these may be added the enlargement of tactile areas and the electrical changes in the muscles which, these authors were the first to demonstrate, precede all others. They point out that the things first and almost universally complained of by those becoming ill are 'a certain numbness in the legs, pain and weight in the legs, and palpitations.' Elsewhere they say (p. 31) of the initial phase of beri-beri : 'The patient first complains of flying pains, of a feeling of weight in the limbs, and that he is quickly fatigued. . . . These symptoms are accompanied by slight modifications of sensibility. Gradually new symptoms appear ; at first a slight œdema along the inner surface of the tibia is met with ; the face takes on a pasty, puffy appearance. . . . At this stage, also, such subjective phenomena as palpitation, increased feeling of weight in the limbs, and depression of spirits appear.' All this time grave dilatation of the heart may be going on, which, giving rise to no symptoms perceptible to the patient, may yet lead to fatal syncope. Next occurs paralysis, followed by atrophy of the muscles affected. Dropsy—*i.e.*, to a marked extent—or general dropsy is in beri-beri, according to these authors, nearly always a sequel of cardiac failing.

'In the diagnosis of beri-beri œdema is only secondary' (p. 59). 'Beri-beri does not commence by œdema round the ankles' (p. 63).

In almost all the cases given for illustration by Pekelharing and Winkler, and in every one of the 38 carefully detailed cases described by Bentley, in which the patient's story of the onset of his disease is related, the earliest signs noted invariably were those of disordered feeling—fatigue, weight, pain, numbness, prickings, pains, cramps, stiffness ; and of weakness, or inability

to use the lower limbs ; *then*, later, swelling, first in legs or feet, then elsewhere. Often the swelling disappeared, while the weakness persisted and grew worse. In many no swelling was ever seen, but there came on the other symptoms, ending in paralysis.

Vomiting at onset, or, indeed, any digestive disturbance, was rarely noted in any of the cases described by these authors. Manson says : ' In ordinary cases the digestion is not seriously affected. . . . Vomiting, unless attributable to ordinary indigestion, is always a grave sign. . . . It is often the precursor of death ' (Davidson, p. 471).

I have recited these well-known, broader features of true tropical beri-beri because, well known and characteristic though they are, their importance, the fact that they are characteristic, has been too often ignored. In the discussion to come, the issue is, in fact, whether a malady which does not possess these its special features shall be considered to be beri-beri.

Among all the symptoms, that which is most easily recognised both by medical man and layman, and that of which easy recognition has probably been chiefly responsible for perpetuation of the confusion of the diseases in which it occurs, is dropsy. Since all dropsies, as effused, resemble each other too closely to afford any criterion of their origin, it is in their mode and site of onset, their time of appearance in the march of a malady, in the signs concomitant with them, that the distinctions must be sought by which they may be referred to a specific source of production.

In ordinary beri-beri, by the time œdema of the scrotum or the shin appears, it will always certainly be found that the nerve-trunks and muscles, especially the peroneal group, are tender and paretic. By the time such œdema has reached any considerable degree—in cases where it progresses—has extensively invaded, say, ankles, feet, legs, and thighs, it may be taken for no less a certainty that there will be considerably advanced paralysis of legs, and perhaps of thigh-extensors, rendering it impossible for the patients to walk or stand without support. Further dropsical progress will be accompanied by more extended paresis, and where there is general anasarca there will almost invariably be also found general grave paresis, if not complete tetraplegia.

Nor in any large number of cases, or groups of cases, of a gravity entailing a fatal issue to many, will it ever fail that some, at least, shall be attacked by the pernicious variety of the disease ; that many shall, without any obvious œdema whatever, pass through the paretic stages to severe paralysis ; and that all shall, in pro-

portion to the extent to which their musculature has suffered, exhibit more or less pronounced—in some cases very marked—atrophy of the muscles.

In short, in tropical epidemics œdema is a symptom developed *pari passu* and as a part of the general neuritis, not preceding nor independent of it. *It never occurs alone.* On the other hand, the other symptoms of true beri-beri, the purely nerve-signs, sensory and motor, do frequently occur alone, or with only such a slight and fugacious œdema (of shin) as often goes quite unnoticed. These are the cases described as 'dry' beri-beri. And whereas, in most epidemics, the 'wet' or severely dropsical cases are not common, the dry cases form almost everywhere the vast majority.

Now, in the reports of beri-beri happening on board ship—and these are very numerous, for such cases have been brought to almost every port, and have been described by observers of almost every nationality—quite another state of affairs is presented. It is exceptional to find cases—an epidemic—of ship beri-beri conforming with the tropical type. Cases in which œdema is accompanied by paralysis leading to atrophy are rarely mentioned; paralysis, or even paresis, without extensive œdema, never.

The Report of the Norwegian Commission on Beri-beri¹ affords a mine of instances, in many of which the victims detail the onset and progress of their illness. In nearly all the burden is the same: a period of malaise, generally with several days of vomiting (this, exceptional in beri-beri, and a very grave sign, was in the ship cases not of bad omen); then pain in the feet; then swelling, beginning in the feet and spreading upwards; next, progressive weakness, loss of appetite, breathlessness, sending the patient to bed; finally, on arrival at port, some amount of paresis—rarely paralysis—supervening late, with other signs of neuritis, is detected. Occasionally these cases died of syncope; sometimes there were cardiac crises, but death seems oftener to have been the result of exhaustion. But once ashore, they all rapidly recover, and no signs are left behind.

In illustration of what has been asserted, the following extracts from reports to the Norwegian Committee may be given:

Report of Captain Paulsen (*Indstilling*, Bilag vii., p. 19):

'The *Fjord* . . . sailed from Moulmein on September 20, 1899, and

¹ 'Indstilling den . . . Komite for åttage . . . af Beri-beri,' etc., Christiania, 1902.

four weeks later beri-beri broke out. The captain, carpenter, and one A.B. were first ill. The master says he first felt pain in his foot, which then began to swell, and then his leg, and before the ship came to Natal nearly all the crew had it. They buried one seaman at sea the day before Port Natal was sighted, and as he was laid on the deck water ran out of his ears, and he became so heavy that it took six men to lift him. When the ship arrived at Cape Town there were only five men on deck; there was only one of the crew that beri-beri did not attack, and that was a coolie boy. One of the seamen died in hospital at Cape Town, and all the rest (twelve seamen) were paid off. The ship remained at Cape Town about a month. The first mate had a slight touch on the passage home from Cape Town. The captain said that if he pushed his finger in his leg the indentation made would remain for about half an hour.'

No mention of anything resembling paralysis.

Report of Captain Tebiason, ship *Hercules* (*Indstilling*, Bilag vii., p. 20):

'After being out about forty days beri-beri broke out, the captain, first mate, and five sailors being struck down. The captain first felt pain in his feet, which gradually grew upwards; he became much swollen—*so much, in fact, that he could not get on his clothes* [from which it appears he was still active]; and if he placed his finger on the leg the mark would show for about twenty to thirty minutes. Upon arrival at Queensborough, he had some good meals on shore, and *after a week was quite well again.*' [Such a result is hardly credible in beri-beri so advanced as the general anasarca would indicate.]

No mention of paralysis.

Report of Captain Nielsen, barque *Defenser* (*Indstilling*, Bilag vii., p. 27):

'Captain and steward only ones affected. . . . The steward after three and a half months at sea complained of shortness of breath, legs swelling, weakness, etc. About two weeks after that—viz., last Thursday, May 18—I felt it come on suddenly.' [Apparently no difference of symptoms.]

No mention of paralysis.

Report of C. Langham, second mate, ship *Sound of Jura* (*Indstilling*, Bilag vii., p. 28):

'I joined the vessel in Callao . . . and sailed thence on October 23. . . . About three weeks before reaching the English Channel I became sick, and could eat nothing. After taking an

emetic I felt better and went again on deck, but had to go below again and lie up. Could eat nothing but a little toast three times a day, and drink a little tea. Then the left leg started to swell from 14 inches to 19 inches in girth; then in two days the right leg began to swell, then the right arm, then the chest. After five days the swellings subsided, then started again. . . . The sailmaker died of beri-beri before we reached Falmouth. He and I were taken ill the same night. . . . The mate showed symptoms before reaching the English Channel. . . .'

No mention of paralysis.

Report of Captain Hansen, schooner *Felix* (*Indstilling*, Bilag lv., p. 151):

'After I had been about thirty days at sea my sight became affected, until I could not distinguish the man at the wheel; my sight has always been very keen, like a cat's. When we were thirty-six days at sea, I felt a tingling under the skin of my legs, as if worms were crawling about. Then the legs began to swell, and the swelling has extended over the whole body to my head and hands. I have never been ill nor had a doctor before. No one of the crew has been affected. I had exactly the same diet as the crew, except that occasionally I had a tin of sardines extra.'

No mention of paralysis.

It must be supposed that sailors, whose livelihood depends upon their activity, would be the first to note loss of power in their muscles, and it is hardly imaginable that a malady so strange to them, yet at the same time so obvious as paralysis, could go unobserved.

Yet that it may not be objected that a conclusion of such importance (as the absence of definite paralysis as a symptom of those ship cases) has been based only upon the untutored observation of simple sailors, and is therefore not reliable, records of precisely similar cases made by medical men may be submitted.

Thus, in the same collection of evidence there appear the following reports:

Report of Dr. Owen, Falmouth, on cases ex-barque *Petrus* (*Indstilling*, Bilag xxxi., p. 89):

'I beg to report that Captain Th. Anderson and three of his crew—viz., the sailmaker, carpenter, and an A.B.—were admitted to the Royal Cornwall Sailors' Home, Falmouth, on June 22, 1896, suffering from beri-beri.

'On admission, Captain Anderson was very ill, his symptoms being as follows: general anasarca, most marked in the lower

extremities; shortness of breath on the slightest exertion; very weak heart's action, occasionally intermittent; anæmia, with at times a good deal of præcordial pain—at other times general muscular pains, chiefly in arms; numbness of lower extremities, which were *nearly* paralyzed (paraplegia); loss of patellar reflexes; cremasteric reflexes present. His digestive system was very much upset, having almost total anorexia and very troublesome constipation, . . . the bowels being distended with large scybala. . . . Loss of sleep; temperature normal; anxious expression; tongue pale, but fairly clean; no scorbutic condition of gums, nor any signs of scurvy elsewhere; lungs healthy.

'The carpenter was suffering from very marked general dropsy, anæmia, intermittent heart's action, and coarse crepitation over bases of both lungs, . . . inability to walk, and slight anorexia.

'The sailmaker and A.B. were only suffering from *inability to walk owing to breathlessness and very marked* general dropsy and anæmia. Urine normal but scanty in all four cases.

'The three last cases all made a rapid recovery under diuretics, diaphoretics, aperients, etc.'

The following are notes of cases treated at the Tropical School, Seamen's Hospital, London (*Indstilling*, p. 119):

'1. Jons Samuelson, under care of Dr. Duncan. Date of illness, September 30; admitted October 30; discharged November 14.

'First taken ill in the middle of September. Lost his appetite and strength; his legs and feet felt numb, and the front of his legs swelled; had a little pain in the back, and *vomited for three days*, nothing remaining in the stomach. Was put upon another vessel, and the change of diet seemed to stop the vomiting. Strength improved a little, but legs got worse. Has been in Seamen's Hospital, Greenwich, one week, and is much better. Passed very little urine on board ship, plenty since in hospital.

'*Present Condition*.—Skin sallow and dry; conjunctiva not quite clear; tongue clean, reddish; appetite good; lungs normal; heart—first sound exaggerated, spacing imperfect, dulness over left ventricle increased, open beat neither seen nor felt; pulsation in right jugular; pulse 80; temperature 97° F.

'Hand-grasp not strong; has foot-drop in walking; slight response to tickling soles; hyperæsthesia of calf muscles; sensation of fronts of tibia dull; very slight knee-jerks; no right.

'October 31.—Twelve ounces urine in sixteen hours.

'November 1.—Twenty ounces urine in twenty-four hours; went out once.

'November 2.—Forty ounces urine.

'November 9.—Able to sit up; gaining strength in limbs and generally.

'November 14.—Discharged at own request.

'2. Otto Mathias, under care of Dr. Duncan. Date of illness, September 18; admitted September 30; discharged October 31.

'About six weeks ago legs began to feel dead. *Vomited five days*. Vomiting ceased when he reached the other ship and got different food; had pain in umbilical region for a week; bowels were constipated; passed plenty of urine; had no palpitation of the heart. His legs swelled in front, pitted on pressure; *was walking about the whole time*, and he says that made him better.

'*Present Condition*.—Tongue clean; appetite good; heart normal; pulse 80.

'Superficial and deep reflexes normal. No hyperæsthesia of calf; walks well; hand-grasp good.

'October 31.—Discharged convalescent.'

'3. H. Theodor Falch, under care of Dr. Duncan. Ill October 5; admitted November 13; discharged December 19.

'Was first taken ill on October 5. Noticed his legs were swollen and tender on the fronts of tibia; ankles not swollen; some days after was swollen in the face; had loss of appetite and strength; felt sick and weak; *vomited for five days*; a little difficulty in walking.

'*Present Condition*.—Temperature normal; tongue clean; appetite good; no œdema; slight pulsation in neck; apex-beat felt; no spacing between equal heart-sound; no murmur; hand-grasp weak; knee reflex absent; anæsthesia of soles and dorsum of feet; tenderness of calf and thigh muscles; walks slowly and deliberately; can stand with eyes shut.

'Urine, 15 ounces in sixteen hours; November 15, 20 ounces. This patient rapidly improved, and was discharged convalescent on December 19.'

'4. Anders Christensen, under care of Dr. Duncan. Ill September 1; admitted November 13; discharged December 19.

'Fell ill at the end of a month, in September. His ankles swelled, and then his legs pitted on pressure; lost strength and appetite, and had to go to bed; passed very little urine; *vomited for about five days*.

'*Present Condition*.—Temperature normal; tongue clean; appetite good; apex-beat seen and felt just below nipple; spacing normal; no murmur; pulse 72.

'Walks with shuffling gait; cannot walk without support, nor stand with eyes closed. Knee reflexes absent; hyperæsthesia of muscles of calves and thighs. No anæsthesia; no œdema. Urine, 36 ounces.

'Discharged convalescent on December 19.'

'5. N. E. Anderson, under care of Dr. Duncan. Ill August 10; admitted November 13; discharged December 19.

'Taken ill on board ship during voyage from Rangoon; felt weak in legs on August 26; noticed his legs were swollen round ankles; this swelling went up legs in front; the swelling pitted on pressure. In three weeks his face and neck were swollen. Had no vomiting till two days before arrival at Falmouth, October 5. The swelling had gone from his legs, but he could not walk without assistance, and had no feeling in legs. Was taken to hospital, and got a little better in his walking there. Nine had the same illness, and one died.

'*Present Condition*.—Skin natural; tongue clean; temperature normal; no œdema; slight pulsation in neck; pulse 80. Apex normal; first sound indistinct. Urine, 5 ounces first sixteen hours. Hand-grasp weak. Anæsthesia over tibia; hyperæsthesia of muscles of thighs. *Knee reflexes exaggerated*. No ankle clonus. Walks only with support; cannot stand with eyes shut; shuffles as he walks, touching the ground with heels and great-toe simultaneously (side-drag).

'November 19.—Urine in fair quantity. Patient gaining strength.

'November 30.—Muscles of extremities atrophied local. Cannot walk without support; with eyes shut, falls backward; gait shuffling; walks by holding on supports, whole foot flat down [not toe-drop]. Moves fairly well in bed. Hand-grasp weak, but getting stronger. Sensation to pin, and pinching absent to elbow on back. Feels pain on soles, but not on dorsum, till it comes to tubercle of tibia. Sensation to touch and temperature same as pain. Plantar reflexes gone. *Knee-jerks increased*.

'December 4.—General improvement; knee reflexes normal.

'December 7.—Steady improvement; can walk alone, and stand with eyes shut.

'December 19.—Discharged convalescent.'

In this patient's history the following note appears: 'In Rangoon had fresh beef and potatoes every day; *bread made from rice flour*. Had rice bread all the passage home; no biscuits, salt pork, salt beef, preserved fish. All the food had a bad taste;

says it was rotten. The other eight on the ship were sick in the same way ; two were more swelled.'

It will be observed that in the last five cases the history, elicited probably by leading questions, approaches in some respects the ordinary one of beri-beri. Numbness in some cases preceded swelling ; the latter, in two cases, began over the tibia. On admission, there were hyperæsthesia of muscles (pain on pressure), and some motor paresis, or paralysis. In (1) there was 'foot-drop' in walking ; (3) could walk 'slowly and deliberately' ; (4) had 'shuffling gait' ; (5) 'shuffles'—dragged his feet sideways along the ground.

Such observations do not, however, make up the picture of extensor paralysis, entailing the marked toe-drop with inverted foot, so familiar in almost every grade in the true paralytic beri-beri.

The true beri-beric posture—the thigh, leg, and foot over-extended in one long backward curve (in which the posterior crucial ligament of the knee seems to be all that saves the arc from collapse) ; the body toppling ever forward, and saved from falling only by a stick ; the widespread limbs, the flail-like leg, the high-stepping gait ; the whole resulting in a characteristic attitude and motion which must strike the least observant of spectators—this is a picture wholly missing from the accounts of the ship beri-berics.

It is notable, too, that even expert observation fails to record in any of the cases *tenderness of nerve-trunks*—invariably present in some degree, according to the author's experience, in all established cases ; in but only one case is there any note of *atrophy*, equally in all advanced cases an invariable finding. Although there is weakness of hand-grasp, there is in no case *wrist-drop*. The signs of cardiac dilatation, almost a normal result of beri-beri, are, except in (1), absent. Finally, there is in each case some positive anomaly. In every one severe vomiting (in three of them for *five* days !) marked the onset. In the last (the one otherwise most resembling true beri-beri, and perhaps really complicated by it) the tendon reflexes were exaggerated at the same time that there was extensive apparent paresis of lower extremities—a condition unmentioned by any author, and never met with by the present writer in a very long experience.

We have therefore described in detail 25 cases, and by reference many more cases of ship beri-beri, and the *Indstilling* contains accounts of very many more such occurring in the course of

epidemics on over one hundred ships. None of the accounts which are detailed present a typical picture of tropical beri-beri; all have œdema. But throughout there is not an indication that any case of paralysis without œdema, or 'dry' beri-beri, the most typical product of the disease, had ever been noted to occur.¹

Not only in the Norwegian Committee's Report, but in the medical journals of almost every sea-bordered country, are to be found accounts of epidemics agreeing with those described. It seems incredible that in none of them should the type of 'dry' atrophic paralysis, so obvious in epidemics on land, have been discriminated, had they been present, or that such cases should not have been present had the disease been true beri-beri.

Judged by this broad and, as it seems to the writer, sufficient criterion, therefore, it is submitted that ship epidemics of the kind, though a form of neuritis, are not that particular form of it which is properly termed beri-beri.

Nocht.—As the result of extensive acquaintance with the ship-malady as met with in the port of Hamburg, and for a reason

¹ Nor is it only in what they lack that the ship cases fail to conform to ordinary beri-beri. In almost all the records symptoms are noted as present which are of great rarity in the real disease.

Thus, Kirchberg (*Gaz. Méd. de Nantes*, December 12, 1893, p. 10) details the case of three Norwegian sailors treated by him in the Nantes Hospital, taken off a ship coming from Guadeloupe. Each of these had, with eight others of the crew, as the first signs of illness great weakness, dropsy, and *disturbance of vision*. Objects at whatever distance seen were blurred and foggy (œdema of fundus? optic neuritis?). Two had also diplopia. Mossé and Destarac (*Rev. de Méd.*, December 10, 1895, vol. xv., No. 12, p. 977), in an exactly similar case (not, however, from a ship), noted optic neuritis, entailing total blindness for several months. These conditions were recovered from.

Norman (*British Medical Journal*, 1899, vol. i., p. 409) refers to two Norwegian sailing-ships returning from Florida, cases from which were admitted to Sir Patrick Dun's Hospital, Dublin, in 1898, and which were described by Dr. Smith at the Royal Academy of Medicine, Ireland, January 27, 1899.

In one case the teeth had fallen out, but this was not attributed to beri-beri. One patient had an extensive series of herpetic patches, which turned into bullæ, and left little ulcers after them. There were three or four cases which exhibited a curious eruption, not distinguished, the reporter said, from *erythema nodosum*. These cases Norman referred to as of the same type of disease then prevalent at the Richmond Asylum, which perhaps they were.

In the cases which Birge (*Boston Medical and Surgical Journal*, 1890, vol. cxxiii., p. 464), Shattuck (*ibid.*, 1881, vol. cv., pp. 400, 577), Putnam (*ibid.*, cxxiii., pp. 62, 244), and Roosefelt (*ibid.*, 1886) describe, among Newfoundland fishermen, the principal signs of neuritis were confined to the hands.

similar to that expressed above, Nocht¹ very emphatically denies its identity with beri-beri. He points out how conspicuous a contrast the *course* of the one disease offers to that of the other, the dropsical ship cases reviving, like scorbutics, from the moment they reach the shore (and get a new diet), and becoming almost always completely well in a few days or weeks ; whereas the true beri-beric progresses only slowly, and often through relapses to recovery.

He considers, as the writer does, the total absence of the 'dry' atrophic forms in the ship-disorder—a convincing proof that the latter cannot be beri-beri. 'Never,' he says, 'have I met among either active or convalescent cases of this ship-incurred disease with the atrophic or 'dry' form of beri-beri ; moreover, never once has any survivor, in describing the symptoms observed at the height of any epidemic during a voyage, depicted this so common form of beri-beri.'²

Nocht considers the ship disease to be essentially a sort of scurvy, and observes that a few years ago no one would have thought of describing cases of swelling and weakness of the limbs, even without any definite gum lesions, occurring in the course of sea-voyages other than as scurvy. The 'abuse' of the word 'beri-beri,' as applied to such cases, arose, he asserts, at two ports—Punta Delgada and Falmouth.

In view of this conclusion, the discovery even of many instances in which an asserted beri-beri has occurred on board ships upon which no rice has been eaten becomes divested, as an objection to our theory, of all importance. None the less, since, concomitantly with the perhaps commoner spurious form of it, true beri-beri also does occur on ships, and the maladies complicate each other, it remains of interest to consider some of the circumstances attending the occurrence of both these diseases.

The Norwegian Committee on Beri-beri.—This Committee, which, under the presidency of Ucherman, lately investigated the circumstances of 104 ships upon which outbreaks occurred, concluded that they were independent of water and of rice, of point of departure or ports of call. They prevailed particularly on long voyages, chiefly in sailing-vessels, and most in Norwegians, and especially where there was inadequate or tinned food. They bore no relation to scurvy, but were stopped by the use of different food, and cases were cured when put on fresh provisions. The disease was considered to be 'due to intoxication by tainted animal or vegetable food.'

¹ *Hansa*, No. 29, 1900.

² *Indstilling*, p. 131.
30

The usual effects of *tainted* food are, however, unlike beri-beri. They are, as a rule, acute gastro-intestinal disturbance, early and profound collapse.

Moreover, it has repeatedly occurred upon ships in the past, as well as under other circumstances, that parties of men have been compelled to maintain themselves on, or even prefer habitually to consume, tainted or actually decomposed food for long periods, and yet no such definite clinical condition as beri-beri has resulted.

The effect, the peculiar syndrome of symptoms simulating beri-beri, which occurs on the ships, although it can thus hardly be ascribed to the consumption of food tainted or decomposed in an ordinary sense, may yet be due to a special form of such change, the result, perhaps, of a specific ptomaine which the conditions peculiar to ship-board life and ship supplies are more apt than any other to promote.

It is on this basis that Ucherman,¹ who fails to make any clinical distinction between the spurious beri-beri of the ships and the tropical disorder, has sought to provide for them a common ground of etiology. He would define beri-beri as a neuritis, caused by a toxin-poisoning derived either from animal or vegetable food, tropical beri-beri being derived from the latter—*i.e.*, from decayed rice—the ship malady from bad preserved foods. He thus regards both clinical types and their specific agents as identical, considering only the sources of *derivation* of the latter to differ.

The author's view is that both the symptoms and the causes of tropical and ship beri-beri are distinct.

Beri-beri is an amyotrophic myelo-neuropathy, the cause of which is a poison secreted by a parasite peculiar to rice.

'Ship beri-beri' is an allied but distinct neuropathy due to the action of some ptomaine developed in tinned foods.

While the etiological factors thus become multiplied, the epidemiology of what has hitherto been classed as beri-beri, and found etiologically inexplicable, is rendered simple.

The ascription of real beri-beri to its true cause in a rice-derived poison, which it has been the object of this work to demonstrate, no longer finds an obstacle in the fact that on ships where no rice is eaten epidemics of a similar disease occur.

First of all, in every such epidemic, it must be shown that the disease more than simulates—it must correspond with the type.

¹ *Zentralbl. f. Inn. Med.*, 1904, vol. xxv., No. 24, p. 617.

Should the outbreak be true beri-beri, and the use of rice directly be disproved, even then the rice factor is not excluded.

For, without distinguishing such epidemics in non-rice-eating crews as a separate form of polyneuritis, and seeking for their source among the fifty accredited causes of that condition, or adding another to their number, it can still be supposed that they are either (1) derived from rice indirectly, through glucose, or through the use of rice to adulterate flour; or (2) from aliments casually invaded by the specific agent which usually affects rice; or (3) may be the result of the beri-beric agent having affected other cereals.

Bullmore's Observations.—Bullmore, whose experience of ship cases, as Surgeon to the Falmouth Hospital, is extensive, propounds the following conclusions:

'1. The beri-beri which reaches us is not the same as the beri-beri of the Malay Peninsula and elsewhere.'¹

'2. The disease . . . is due to an alkaloidal poisoning fanned into flame by the disarranged digestion of sailors who are kept on improper diet.'²

The source of the alkaloids he considers to be the tinned and salted foods, the liability of which to decompose and produce ptomaines during long voyages through tropical weather, he insists, must be great. He pertinently says: 'Pekelharing says' (of true beri-beri) 'it is due to a diplococcus acting like muscarine. Why not muscarine, or one of its allies?'

In his view, therefore, the ship disorder is a ptomaine-poisoning and a species of auto-intoxication combined. In the former part of this belief the writer believes Bullmore to have nearly touched the issue. For a disease so peculiar to ship-life, conditions peculiar to ship-life must afford the reason. But, except in the matter of dietary, is there any possible efficient morbid agency peculiar to the sailor?

To unwholesome articles of food, then, there can be little doubt, as Ucherman and Bullmore agree, the cause of the dropsical affection—pseudo-scorbutus or pseudo-beri-beri, whichever it be—is to be imputed. The particular form of poison, the particular fault of preparation to which its formation in such stores is due, remain for the chemist to determine.

Bullmore, like Ucherman, has drawn attention to the special liability of sailing-ships and of Norwegian crews to beri-beri. Of forty-three ships putting in at Falmouth between 1896 and 1901

¹ *Indstilling*, p. 126. ² *Ibid.*, p. 129; also *Lancet*, September 22, 1900.

468 THE CAUSE AND PREVENTION OF BERI-BERI

upon which there had been beri-beri, not one was a steamer. The proportion per cent., according to different flags, of all craft calling at Falmouth, and of the beri-beri-stricken ones, was as follows :

	British.	Norwegian.	German.	Swedes.	Others.	Total.
All ships calling (429)	48	18	14	4	15	99
Beri-beri ships (48)	7	65	9	7	12	100

The proportion of sailing to other craft carrying different flags and not having beri-beri is not given. It is not clear, therefore, from Bullmore's statements,¹ whether the greater liability of the Norwegian sailing-ships to carry the disease is owing merely to their being sailers, or because they are Norwegians. The factors determining special incidence on sailing-vessels are no doubt that they are bound for longer voyages, and touch less frequently at ports where provisions can be renewed. If the proclivity of *Norwegians*, especially among all sailing-ships, be a fact, it points to some national characteristic affecting diet as determining the result.

Bullmore, who carefully investigated the circumstances of his forty-eight ships, gives the following as an average scale of diet on a Norwegian vessel :

Sunday.—Fresh meat, dough, sweet soup, ship biscuit.

Monday.—Pea-soup, fresh vegetables, salt beef, fresh bread, ship biscuit.

Tuesday.—Sweet soup, stock fish, tinned cabbage and potatoes, ship biscuit.

Wednesday.—Salt beef, fresh bread, tinned cabbage, ship biscuit.

¹ I find from analysis of figures from Lloyd's Register that among the sailing-craft of the world Norwegian vessels do preponderate as compared with other European nations (not British), and that among all Norwegian craft the proportion of sailing-vessels is greater than in the case among other flags. The following is the exact analysis of the number of craft of over 100 tons possessed by all nationalities in 1903. Total vessels over 100 tons, 29,091 :

Distribution.	British.	Norwegian.	German.	Danish.	All Others.	Total.
Sailing-ships ...	95	49	17	15	256	432
Steamers ...	279	29	44	13	203	568
Total ...	374	78	61	28	459	1,000

Thursday.—Same as on Sunday, ship biscuit.

Friday.—Pea-soup, fresh vegetables, pork, ship biscuit.

Saturday.—Sweet soup, stock fish, tinned potatoes, ship biscuit.

The 'sweet soup' is made with dried apples or prunes. Bullmore adds that 'there was always given bread with every meal, generally in the form of dried biscuit.' *The biscuit* was kept in barrels or large iron tanks. It was '*nearly always found weevily or mouldy on opening*.' The flour, Bullmore states, stored in similar receptacles, kept well. But seeing how rice, quite dry and sound and sweet on inspection, proves toxic, the flour may have been in a similar condition.

The fact, which may be of significance, was also brought out by Bullmore that on such ships the *captains* are rather more liable than their crews to the disease.¹ Since, on sixteen ships affected, among 295 men, representing the total crews, there were 106 cases, or 39.3 per cent., while of the sixteen captains, nine, or 56.2 per cent. suffered.²

Bullmore suggests that the reason is because the captains were 'more exposed.' But a likelier one would seem to be that (the cause lying in diet) they had more of the injurious articles than the men. To a landsman the Norwegian ships' dietary seems far from appetizing, plausibly suggestive of unwholesomeness and disease. Nevertheless, it is certain that in earlier days, when ships of all flags were sailers, the food was far less varied, and more often more unwholesome, and voyages were of longer duration, and yet beri-beri was not prevalent. Scurvy was well known, and its ravages are frequently recorded; but although such a combination as dropsy and paralysis could not escape notice or fail to be distinguished as a malady, the current medical literature of the time never records its occurrence.³

On the other hand, of late years, as Ucherman, too, has observed, the disease has become in all parts of the world increasingly frequent.

Moreover, fluctuations in its prevalence at sea, so far as one

¹ According to Ucherman's results also, captains and officers are far more liable than the crews.

² Excluding deaths from violence, the rate of mortality from beri-beri among the men of the merchant shipping during 1900 was 18 *per* 1,000 of *all deaths*. Sixty actual deaths from this cause at home and foreign ports were registered.

³ The writer at least has searched with some care the files of three such periodicals for the last sixty years, and failed to find any record of beri-beri on ships (other than references to the Japanese and Dutch East India navies from 1875 onwards) prior to 1884.

can judge by partial records, appear to agree with those in its endemic areas, and correspond with periods of greater or less frequency of outbreaks on land in new places. Thus, of six vessels of the fleet to which Rees has drawn attention as special 'beri-beri ships,' there was an outbreak in 1890 upon only one. In this year there was also general decline of beri-beri in the hospitals of the Malay States.

In the four following years (1891-1894) outbreaks happened on five or six ships in each year, but only on one in 1895, and one again in 1897. There were three in 1896. Now, these years of decrease or increase on the ships coincided again with similar variations of prevalence in the Malay hospitals, in the Dutch colonies, and even in the now nearly purged Japanese fleets, and other places. In 1895, the year of least activity on the ships, the outbreak at the Dublin Asylum, which had begun the year before, ceased, recurring in 1896; 1897 was also a period of commencing fall in nearly all the Malay hospitals.

All the forty-eight ships mentioned by Bullmore came in between 1896 and 1901. The year of least frequency was 1894; that in which they were most numerous 1900. This latter was one of general decline in the Malay States; but in the year preceding, 1899, there was a wave of increase in twenty out of twenty-seven stations observed.

In this periodic movement (if it be real) *all* the cases, both true and apparent beri-beri, are referred to together. But it is likely that the element which fluctuates chiefly must be the true disorder, of which perhaps the greater part of the asserted cases are then examples.

Such correspondence (which it is probable that wider investigation would show to be even closer) between beri-beri at sea and beri-beri in its endemic centres strengthens the presumption that the cause in each is ultimately derived from identical sources. The increasing spread of the disease in late years, inexplicable on the mere ground of increased traffic (since there is no evidence of infection), must be due to economic reasons. Among these the cheapening of ocean freights, which has everywhere promoted the introduction of Eastern products, enabling rice especially to be sold at prices lower than other cereals, has led to a greatly increased importation of that grain. It is at least probable that in ships the flour served as wheaten is often largely adulterated with rice.

If the liability especially of *Norwegian* vessels to beri-beri be

not apparent merely, simple adulteration of biscuit, etc., with rice will hardly explain the fact, for the adulterator owns no nationality. For such peculiar racial incidence a racial origin would have to be sought. Since there is no race immunity, this must lie in the *source* whence the food is obtained. The indication is that the cereals produced or used in Norway contain a poison which those of other countries do not. It may be that the Norwegian owner, for some reason or other, is more wont to use rice-adulterated flour; it is certain that, as compared with other vessels, the Norwegian vessels are the more cheaply provided. But if this explanation fail, the poison must lie in cereals other than rice, or secondary products derived from them. This would agree with the theory already elaborated, and is in accord with the analogy of ergotism.

To sum up in brief the considerations applying to the beri-beri of ships and asylums, the only known places of its occurrence in temperate latitudes :

1. The described epidemics in European and American asylums are not typical, in the writer's opinion not true, beri-beri, though it is possible some may have been outbreaks of it in a modified form.
2. On ships a form of neuritis, ascribable to ptomaine-poisoning, also occurs, complicating and complicated by epidemics of true beri-beri.
3. Often it is likely these epidemics are due to rice equally with the cases of the tropics, but that agent has appeared to be excluded, because operating indirectly through channels which have not been searched.
4. Where rice can with certainty be excluded, the agent which causes beri-beri may have infected other aliments which have been stored in contact with rice; or other cereals may have been at fault, derived from crops affected by the same parasite. The available evidence goes to show at least that the food-supply in all the epidemics either has with certainty, or may have been, inferior.
5. The occurrence of these epidemics, wholly isolated from each other, and under circumstances which no data of infection, no conceptions of miasm, can explain, amid populations using apparently identical food, but remaining unaffected, is the result, not of the *cause* being exceptionally applied, but of exceptional response to a cause which is widely prevalent.

The influence is *common*, but the power to resist it unequally

distributed; so that though, as in ergotism, all are perhaps eating similar amounts of one and the same poison, those whose condition gives them evil pre-eminence in susceptibility to the disease first succumb.

Ships and asylums so circumstanced in this way afford a sort of barometric index of the presence of the 'beri-genic' agent.

Their especial liability to the disease should afford reason for more especial care in the provision of food, by liberal and wholesome supplies of which there can be little doubt the disease may be prevented.

The paradoxes and ambiguities, the many anomalies in the natural history of true beri-beri, are, therefore, simply enough explained when the disease is regarded as a grain-intoxication.

The fact that sometimes at sea, and occasionally on land (even, in rare instances, in the tropics), the exact derivation of the poison has not been traced is no bar to the acceptance of this view.

Since, if the disease described as beri-beri in all quarters is identical, it is worth while repeating, the same causal agent must be everywhere at work: if its vehicle or medium in the greater number of instances be grain, it is at least likely to be of a similar character in all. The presumption is one to which force is lent by the nature of the evidence, extraordinarily complete and extensive, by which the connection of the disease has been proved with at least one sort of cereal.

By the strictest logical method, by rigorous examination of all the facts available as evidence, it has been demonstrated that in the vast majority of cases of beri-beri all over the world the cause certainly lies in rice; while in a large proportion of the scanty minority of instances the same factor has not been excluded, because various channels through which it might have operated, but indirectly, have not been examined.

The ninety-and-nine cases being thus traced undoubtedly to rice, it were perfectly legitimate to refuse to admit under the same definition the exception in which rice plays no part. But the conception of the disease as one marked unity, under whatever circumstances occurring, would thereby be destroyed; while medicine affords numerous analogies, where the same toxic agent is distributed through various but similar media. *Beri-beri, therefore, it may be accepted, is always a grain-intoxication.*

NOTE ON THE RELATIONS BETWEEN BERI-BERI AND ARSENIC.

It will not have escaped notice that *glucose*, if possibly a medium of beri-beri intoxication, is, as a vehicle also at times of arsenic, likely to entail the consequences of that drug, and so complicate the clinical symptoms of beri-beri. Clouet,¹ of Rouen, in 1877 drew attention to the presence of arsenic in commercial glucose. Samples analyzed by him showed from 0.005 to 0.007 gramme per kilo.

Delépine² states that he has found in specimens of glucose used in brewing as much as 4 to 6½ grains of arsenious acid per pound. Supposing a jam or 'golden syrup' to contain 25 per cent. of such a glucose as the last, and 2 ounces of it be given at each of two meals—a common allowance in schools (or asylums)—it would result that the consumer would be taking two-fifths of a grain of arsenic daily.

Various inflammatory and paralytic lesions may follow the consumption of arsenic in quantities much smaller than this. Naunyn says that 'doses of less than $\frac{1}{18}$ grain have sometimes produced injurious effects on adults. Evident signs of poisoning have been observed as the result of taking $\frac{1}{4}$ to $\frac{1}{2}$ grain. In one case alarming symptoms were produced in a family by the continued use during four days of $\frac{1}{18}$ grain.'

In the epidemic among beer-drinkers described by Reynolds³ the beer in use contained from $\frac{1}{2}$ grain to 3 grains per gallon; and many subjects certainly showed signs of poisoning who were taking only two or three glasses per diem (from $\frac{1}{8}$ to $\frac{3}{4}$ grain). Reynolds states that 'in susceptible subjects the mere application of arsenic plasters to the skin will produce *the most profound neuritis, as I have myself seen.*' Although the writer can find in the literature no parallel to an occurrence such as this, it is evident enough that quite small doses of arsenic will entail decided toxic effects.

There is a very high probability, therefore, that in epidemics

¹ 'Annales d'Hygiène,' pub. 1878, p. 145.

² Report on Samples of Beer, Brewing and other Materials, Salford, 1901.

³ *British Medical Journal*, November 24, 1900.

of beri-beri indirectly produced through glucose, signs of arsenic poisoning will be occasionally superadded.

In the epidemic occurring at the Richmond Asylum a few signs were noted in some of the cases of a character certainly unusual in beri-beri, and suggestive of complication with arsenic. Thus: 'A few cases were preceded by vomiting and sometimes diarrhoea; but this concurrence was rare.' 'Tendency to *contractures* appeared in half a dozen cases . . . only two patients have remained crippled from this condition.'¹

With this may be compared the experience of Dixon Mann² in the beer-drinkers' epidemic, who stated that 'along with the tenderness a spastic condition of the muscles was usually present, the legs being fixed on the thighs so rigidly that they could not be straightened.'

There were 'patches of hypo-æsthesia surrounded by hyper-æsthesia often'—*anæsthesia dolorosa*. 'The superficial nerves were tender, in some cases extremely so.' 'In one case there was unilateral paralysis of the third nerve.'³

Such symptoms are seldom met with in any form of beri-beri, and probably never happen in uncomplicated cases. They are, on the contrary, frequent and, indeed, characteristic of arsenical neuritis.

Ross and Reynolds⁴ have referred to the great similarity of beri-beri to alcoholic and arsenical neuritis. One of them (Ross) states that having seen cases of arsenical paralysis during the epidemic of neuritis among beer-drinkers at Chester, he was so struck with their likeness to the cases of beri-beri seen in the East that he was led to suggest that some of the tropical cases of beri-beri might really be of the same nature. The two observers report an interesting case of 'arsenical' neuritis, which was held by several to be probably beri-beri, in which the etiology was further complicated by pregnancy. The patient was a total abstainer brought from Sierra Leone, living while out there largely on *canned fruits*, from glucose contained in which Ross suggested the arsenic might have been derived. But analyses of many similar samples subsequently showed their freedom from it, at least in poisonous quantities.

The possibility of mistaking arsenical neuritis for beri-beri

¹ Worman, *loc. cit.*

² *British Medical Journal*, October 12, 1901, vol. ii., p. 1044.

³ Worman, *Dublin Journal of Medical Science*, January, 1900, p. 1.

⁴ *British Medical Journal*, 1901, vol. ii., 979.

implies also that of mistaking beri-beri for the former condition. There is nothing in favour of Ross's suggestion that tropical beri-beri is often, if ever, really arsenical poisoning. But it becomes an interesting speculation whether some of the cases set down to arsenic, alcohol, and even other causes of neuritis, may not more probably be assigned to beri-beri. The likelihood of this has already been pointed out by Manson,¹ who states that in his belief 'many of the cases of so-called peripheral neuritis in Britain are really beri-beri.'

In this place the subject cannot be discussed at length, but allusion may be made to the many facts which serve to show that the probability is more than academic. These are: (1) The impossibility of distinguishing with certainty between polyneuritis, poliomyelitis, Landry's paralysis, and other acute ascending palsies; or between some of the best defined of the forms of polyneuritis—*e.g.*, those ascribed to alcohol and to arsenic; and the similarity of beri-beri both to these and the spinal disorders; (2) the entire want of relation between the quantity of dose in forms of toxic neuritis and the result—the disproportion of cause and effect; (3) their occurrence often without any other of those symptoms which are usually associated with the specific cause; and (4) the number and extreme diversity of the various agencies which have been held to produce one and the same clinical result.

The Epidemy of Neuritis among Beer-Drinkers.—In the great epidemy of peripheral neuritis among beer-drinkers which happened in the North of England in the autumn of 1900 the relation of arsenic to which was investigated by a Royal Commission, the whole of the symptoms were ascribed with great confidence to that agent.

Very numerous cases of arsenic poisoning occurred, the symptoms of which were the unequivocal ones—conjunctival irritation, gastric disturbance, desquamation, keratosis, hyperidrosis, characteristic pigmentation, and other skin troubles—specific of that drug. But these were not always accompanied by paralytic symptoms, nor, when the latter were present, even in greatest degree, were they always accompanied by the other and truly specific signs of arsenic action. The ascription of the neuritis to arsenic in these cases is therefore at least open to question, and it is quite possible that the epidemy may have

¹ British Medical Association, July 28, 1898. In *Lancet*, August 6, 1898, ii. 327.

been really one of beri-beri, or even beri-beri combined with pellagra which arsenic merely complicated.

Major Ross¹ drew the attention of the Commission to the close correspondence between beri-beri and the peripheral neuritis attributed to arsenic, and the Commission in consequence took some trouble to collect information as to the possible explanation of tropical ship beri-beri as being itself possibly nothing more than arsenical neuritis. It is singular that it should have occurred neither to Major Ross nor the Commission to suppose that possibly the two terms of assumed identity might be transposed—that, in other words, the neuritis ascribed to arsenic might be beri-beri.

The evidence upon which the causal relation of arsenic to the paralysis in these and other cases has been affirmed is far from conclusive. Since Gautier,² and more recently Bertrand,³ and others, have shown that arsenic is, if not a normal constituent of almost all the tissues of almost all animals, at least invariably present in them, and in more than 'traces,' the detection of the element in the surroundings, the food, the hair, the excreta, even the tissues of individuals, proves practically nothing.

Putnam⁴ affirms that arsenic is to be found in the urine of 30 per cent. of normal healthy persons.

Moreover, arsenic may be, and is frequently, purposely consumed by persons, healthy or sick, in much larger doses than those to which the many peripheral neuritis cases in the epidemic were attributed.

The strongest possible presumption—one almost amounting to a demonstration—that the paralysis could not have been due to the arsenic taken by the beer-drinkers, was afforded by the evidence of Legge,⁵ given before the same Commission, who tabulated eighteen cases of arsenic poisoning acquired industrially by workers engaged in arsenic manufacture, *in none of which was there any sign of neuritis*, although all had marked pigmentation, uvular congestion, hoarseness, and most of them conjunctivitis, and although these signs had been observed in most of them for many years. Reynolds⁶ seeks to explain this admitted inconsistency by supposing that arsenic taken in conjunction

¹ Report of Royal Commission on Arsenical Poisoning, 1903.

² *Comptes Rend. de l'Acad. des Sc.*, cxxix. 929 (1899), and cxxx. 284 (1900).

³ *Ann. de l'Inst. Past.*, t. xvi., No. 8 (August, 1902).

⁴ *Boston Medical and Surgical Journal*, 1888.

⁵ *Report*, *loc. cit.*

⁶ *Loc. cit.*

with alcohol is more potent in its effects, the effects of each towards production of neuritis being combined. But for this there is no more evidence than an observation of Osler's¹ that arsenic was more likely to produce *its specific effects* when given in alcohol than alone. The question being, however, whether neuritis of the form met with in the beer-drinkers, and which was declared exactly to resemble that of alcohol, is one of the specific effects of arsenic, this observation can not be admitted. He also cites Oppenheim's² observation that metal-workers who are alcoholics suffer more from the diseases due to metals than do others. But the 'alcoholic' of Oppenheim is the confirmed and chronic drinker. In many of the beer-neuritis cases it was admitted by Reynolds that the total quantity of beer taken was small—not more than two or three glasses daily—so that neither the quantity of arsenic nor of alcohol consumed could have been large—certainly not such a dose as is usually conceived to be toxic. It was admitted by all who took part in the discussion³ on this epidemy that *alcohol* alone could not be held accountable for the effects, the quantity taken by most of the cases being far less than is taken daily by numbers without ill-effect of any kind. Reynolds himself was at special pains to make his own conviction clear that nearly all the cases of peripheral neuritis previously ascribed to alcohol were probably due to arsenic.

It has long been observed, in regard to alcoholic neuritis, that those most apt to suffer from it are not those who consume most alcohol, that in some cases the quantity actually taken has been too little even to produce transitory intoxication, and that this particular lesion often appears alone, without any of the other usual, and undoubtedly specific, effects of the drug.

In regard to both alcohol and arsenic there is thus the paradox, that the effect bears no proportion to the dose; that the most serious of their effects appear (comparatively) oftenest when comparatively least of them is taken; and that healthy adults succumb to doses which are constantly administered with entire impunity even to the sickly and children.

Paradoxes seldom represent facts. The explanation in this case would seem to be that probably the ascription to two causes so diverse as arsenic and alcohol, of a condition clinically indistinguishable in both cases, is in both equally erroneous. It is likely that neither pure alcohol nor pure arsenic produces peri-

¹ Quoted by Reynolds, *loc. cit.*

² *Loc. cit.*

³ *British Medical Journal*, 1901, vol. ii., p. 1044 (October 12).

478 THE CAUSE AND PREVENTION OF BERI-BERI

pheral neuritis, but that this, when observed, has been due to another totally different factor, which might very well be an agent allied to, if not identical with, that which causes beri-beri.

The unlikelihood of pure arsenic and pure alcohol being producers of peripheral neuritis is made stronger by the observation that although animals are subject enough to neuritis, it has not been found possible to produce this condition in any of them experimentally by prolonged dosation with either of these drugs, even in quantities which are ultimately lethal.

In the fact that thousands of persons were attacked suddenly with peripheral neuritis exactly resembling beri-beri, who happened to be at the same time also attacked, many of them by arsenic poisoning, we have exactly the state of affairs which has been suggested as likely to happen when an epidemic of beri-beri should occur, the source of which should be glucose made from any beri-beri-affected sort of corn. In the beer-drinkers' epidemic the source of the illness of the majority, if not of all, of the cases was certainly traced to the use of beer brewed with glucose made from maize and tapioca-flour supplied by a particular firm, which also contained much arsenic.

Does this justify the assertion that the disease was entirely due to arsenic, and not rather to other poisons contained in the same glucose—that it was not, in short, a grain-intoxication, depending ultimately upon the same cause as beri-beri?

SECTION IX

THE PREVENTION OF BERI-BERI

FEW diseases are attended with the good fortune that the demonstration of their cause is at the same time the indication of the remedy. But this may be said to be the case with beri-beri.

For, speaking generally, it may be said that all that is needful is to secure a liberal and wholesome dietary, to avoid the use of rice or its extracts, and the disease will not appear.

Where rice cannot be abandoned, where it is the staple food, all that is necessary is to avoid the use of uncured (stale cleaned white) rice, to eat only the fresh or cured sort, and freedom from the disease will be equally secured.

It is true that in a certain proportion—*i.e.*, when it forms not more than one-third of the total bulk of food eaten—even toxic rice has been taken with impunity ; and in prisons and public institutions, were it a *necessity* to use stale uncured, instead of other sorts of rice, its deleterious action might thus be counteracted by largely diluting it with other food. Such necessity need, however, rarely arise, since both fresh and cured are as easily obtained as stale rice.

But employers generally could not be compelled, often could not, indeed, afford to provide the additional articles of food required to dilute toxic rice to this extent, and it is certain that coolies, badly paid as sinkhehs are, would not do so.

The only practical way, therefore, to prevent beri-beri in a community, is for the State to prohibit the sale of any rice which is not either freshly made, or which has not been 'cured' by boiling or heating in the husk before stripping it.

There would be no difficulty in adopting the latter measure ; it is already carried out on a large scale in several quarters.

Simple as these remedies are, nothing more is wanted, as nothing less will suffice, to banish from the countries which it now infests the blight of beri-beri.

1

APPENDIX I

NOTE ON TRAVERS' SELANGOR GAOLS EXPERIMENT AS TO THE EFFECT OF DIET ON BERI-BERI

AN experiment was made by Dr. E. A. O. Travers as to the dependence of beri-beri on food, including rice, in the Selangor Prisons during 1895-1896. No publication of the results seems to have been made at the time, but an account of them was printed by him in 1891 in reply to a memorandum addressed by the writer of this paper to the Local Government, urging that the cause of beri-beri lay in rice. Travers' account appeared in the *Journal of Tropical Medicine* for August 1, 1901. I quote the main part of it. It was stated—

'In the month of August, 1895, the prisoners in the Pudoh Gaol were severely attacked with beri-beri, which has since caused a large number of deaths, and has continued, with slight occasional intermissions, up to the present time.' 'The Pudoh Gaol was occupied in January, 1895, previous to which all prisoners were confined in the Old Gaol, Kuala Lumpur, about a mile and a half distant. While in the Old Gaol none of the prisoners contracted beri-beri, although they were fed on the same kind of rice procured from the same source as that given them when in the Pudoh Gaol.'

In a table it is then shown that from 1892 to 1894 only nine¹ cases of beri-beri were treated in the prison, all of which were imported, whereas in 1895 there occurred 158 cases; in 1896, 478 cases; and in 1897, 275 cases. *All these cases*, it is stated in the table, were '*contracted in gaol*.'

'It is evident, therefore,' says Travers, 'that in this case the outbreak of beri-beri occurred immediately after a change in the location of the prisoners, the supply of rice remaining the same.' 'The case-mortality in the Pudoh Gaol Infirmary being extremely high, and in view of the apparent influence of locality on the disease, all patients suffering from beri-beri were transferred to the Old Gaol on October 1, 1896.' The result of this arrangement is shown as follows:

¹ In a later statement eighteen (*vide infra*).

482 THE CAUSE AND PREVENTION OF BERI-BERI

Month.	Cases admitted.	Deaths per Cent.	Remarks.
September	32	31·7	Treated in the Pudoh Gaol
October	35	15·5	Treated in the Old Gaol.
November	23	4·15	
December	25	4·25	

'The food for the beri-beri cases treated in the Old Gaol was not only similar in every respect to that used in the Pudoh Gaol, but was actually cooked in the Pudoh Gaol with the food for the other inmates, and was conveyed in a hand-cart to the Old Gaol.

'Encouraged by the evidently beneficial results of change of locality on the sick prisoners, arrangements were made to experiment still further in this direction. On October 21, 1895, sixty prisoners, showing no signs of beri-beri and in apparently good health, were transferred from the Pudoh Gaol to the Old Gaol; from this date to July, 1896, a large gang of prisoners were confined in the Old Gaol.

'The monthly average number of prisoners in each gaol during this period, with the number of fresh cases of beri-beri occurring among them, is shown by the following figures :

Month.	Average Number of Prisoners in Pudoh Gaol.	Number of Beri-beri Cases occurring in Pudoh Gaol.	Average Number of Prisoners in Old Gaol.	Number of Beri-beri Cases in Old Gaol.	
1895 {	October ..	297	38	72	nil
	November ..	337	21	119	"
	December ..	271	28	128	"
1896 {	January ..	280	29	133	"
	February ..	286	47	130	"
	March ..	271	43	119	"
	April ..	275	42	97	"
	May ..	299	36	106	"
	June ..	287	39	84	"

'From October 1 to December 14, 1895, all food supplied to the healthy prisoners in the Old Gaol, as well to the beri-beri patients transferred from the Pudoh Gaol, was, as has been stated above, cooked in the Pudoh Gaol, with the food for the other prisoners, and carried to the Old Gaol twice daily, the diet being exactly the same at both gaols, and the rice being taken out of the same bag and cooked in the same steamer.

'After December 14, 1895, the rations for the prisoners in the Old Gaol were cooked in that institution, raw rations, with the exception of rice, being sent daily from the Pudoh Gaol. The rice was sent direct from the contractor to the Old Gaol, where it was kept in bags, and used as required. It may, I think, be fairly claimed that the opportunity for experiment with regard to the influence of food-supply on the etiology of beri-beri presented by the outbreak at the Pudoh Gaol has been an exceptional one, and that full advantage has been taken of it.

'The results of these experiments and observations seem to indicate

very clearly that in, at any rate, this instance, there was no connection of any kind between the outbreak of beri-beri and the food-supply.'

Such is the author's account of an experiment, the publication of which Manson¹ declared to 'mark an epoch in our knowledge of beri-beri,' and to 'completely refute' the theory of the production of the disease through rice, or, indeed, food of any kind.

At first sight the experiment certainly seems to justify such a conclusion. But it may be shown, without contesting Travers' statements of fact or his figures, but supplementing them with others equally authoritative, being derived, indeed, from official reports made by himself or his colleagues, that the facts may be differently interpreted and yield quite opposite conclusions.

The use in the memorandum by Travers of the expressions, '*while in the Old Gaol none of the prisoners contracted beri-beri*,' '*the beneficial results of the change of locality on the sick prisoners*,' and '*in view of the apparent influence of locality on the disease*,' make it abundantly clear that, in the author's view, there was to be ascribed to the New (Pudoh) Gaol, *qua* site merely, some factor, some influence pathogenic in the direction of beri-beri, from which the other, the Old Gaol site, was free.

The statements that '*the Pudoh Gaol was occupied in January, 1895, previous to which all prisoners were confined to the Old Gaol*,' and that '*it is evident that in this case the outbreak of beri-beri occurred immediately after the change in the location of the prisoners*,' emphasize this conclusion, and leave it no less clearly to be understood that at a given date a wholesale and complete transfer of all prisoners was effected to a site previously unoccupied, and that this transfer was the determining factor in an outbreak of beri-beri, occurring '*immediately*' afterwards.

Yet records to be cited shortly will show: that, so far from the Old Gaol being free from whatever may be supposed to be the '*berigenic*' influence, beri-beri had certainly occurred there in the years prior to those which Travers mentions, while it became severely prevalent in the same place during the years immediately following them, when the gaol had been converted into an asylum; that if continuous occupation of a site by numbers of susceptible persons, who shall, nevertheless, remain even for a long period free of beri-beri, be any test of the absence from it of the pathogenic factor of the malady, then that character ought certainly to be ascribed to the *New (Pudoh) Gaol site, since it was occupied by some 100 convicts continuously for the four and a half years immediately preceding the famous experiment*; that, in any case, little or no influence can be ascribed to the effect of change of locality in producing the outbreak, *since no such abrupt and wholesale transfer of the prisoners from one gaol to the other, as that to which Travers ascribes it, did, in point of fact, take place*; that, finally, it is even open

¹ Meeting British Medical Association, July 30, 1902; reference, *Journal of Tropical Medicine*, August 1, 1902.

484 THE CAUSE AND PREVENTION OF BERI-BERI

to question whether any outbreak at all, in the sense of an epidemic limited to, and produced by causes operating entirely within the prison itself, ever occurred.

Travers' table showing the cases of beri-beri recorded as occurring in the Old Gaol begins in 1892, when only six cases were treated, which were 'not contracted' in that institution. But the Annual Report to Government of the State Surgeon for 1890 says: 'During the year there were 19 cases of beri-beri treated in the prisons, with a case mortality of 10 per cent., a considerable decrease from 1889, when 44 cases were treated, with a case-mortality of 15 per cent. *The gaol return at Kuala Lumpur* showed 14 cases of beri-beri, with 2 deaths. Travers has since favoured me with a record of the cases of beri-beri occurring in the Old Gaol from 1883 onward. They were as follows:

Record of Patients with Beri-beri and Other Diseases at Kuala Lumpur Gaols.

Year.	Total Number of Inpatients.	Cases of Beri-beri.	Deaths from Beri-beri.
1883	342	36	2
1884	326	12	1
1885	215	3	—
1886	243	nil	—
1887	410	11	1
1888	363	12	—
1889	415	26	6
1890	270	14	2
1891	245	9	—
1892	345	10	—
1893	507	4	1
1894	516	4	—
1895	1,082	152	31
1896	1,026	499	47
1897	686	297	55
1898	489	68	2
1899	507	81	7
1900	610	203	7
1901	774	216	6
1902	1,169	901	17

At Old Gaol.

At New Gaol.

Beri-beri certainly occurred, then, in the Old Gaol—was, in fact, the table shows, almost constantly present there to a considerable extent in the years anterior to those cited by Travers.

It became very much more prevalent there immediately after the experiment. After the occupation of the New (Pudoh) Prison, the Old Gaol was converted into a lunatic asylum, and occupied as such on July 2, 1898. In his Report for that year as State Surgeon, Travers states that *shortly after the asylum had been occupied beri-beri broke out.* 'This

is remarkable, as previous to its being converted into an asylum no case of beri-beri was originated among the prisoners in gaol. It would appear that the beri-beri had been imported into the asylum by the Singapore lunatics' [although the constant drafting of all the beri-beric sick prisoners from Pudoh Gaol into the same building had had no such effect upon other prisoners confined in it]. But he adds, '*Many new cases of beri-beri originated in the asylum among other inmates*' not transferred from Singapore.

The total number of inmates, the number of cases of beri-beri, and the mortality from it among them, appear as follows :

Beri-beri in the Kuala Lumpur Asylum.

	1898.	1899.	1900.	1901.	1902.
Total inmates	157	169	166	194	178
Cases of beri-beri	— ¹	67	65	38	21
Deaths from beri-beri ..	— ¹	23	15	27	9

Nothing in the history of this Old Gaol site or building, therefore, justified its being regarded as free from beri-beri, as being a place at which, for whatever reason, beri-beri could not be acquired. Some other explanation than that which Travers adopts must be sought, therefore, for the amelioration of the beri-beri cases who were transferred there from the Old Gaol, which he ascribes directly to change of locality.

Now, alterations in case-mortality as abrupt and remarkable as those instanced to prove the benefit attending removal from the New to the Old Gaol are to be paralleled in the returns of almost every hospital, and are, indeed, a commonplace of observation in beri-beri.

For instance, at Pudoh Gaol itself, at a later date than that of the experiment (1898), Dr. McClosky relates how, in October of that year, of 35 cases treated, 8 died (12·05 per cent.); in November there were 51 admissions and 16 deaths (19·02 per cent.); but in December only 17 cases were admitted, and no deaths occurred.

For any just comparison of the effect of the two different gaols upon the course of the disease, the treatment of a parallel series of cases *simultaneously* at either place could alone afford a sound basis.

But if the history of the Old Gaol afford no reason for regarding it as being, *as a site*, free from whatever makes for beri-beri, that of the New (Pudoh) Gaol, on the other hand, so far as it is known, shows it to have really merited that character. For from 1891 until January 1, 1895, it was constantly occupied by convicts, who laboured there by day and slept there at night. In every possible mode in which *soil* (long the fancied nidus of the disease) could be disturbed, the soil of the Pudoh site was disturbed during that period. Deep foundations for buildings

¹ No record, but 'many new cases originated.'

and wall-courses, shallow excavations for drains, and levelling for yards were continually in process of being made. Throughout these four years the prisoners slept on plank beds upon the ground, in temporary wards of wood, thatched with palm-leaves (attaps), and generally were far less well-housed, and more exposed to damp, than afterwards.

Yet no beri-beri appeared at this time, and whatever may have been the agency determining its appearance at the same site later (August, 1895), when the full strength of the prisoners had come into residence, it cannot be conceived, on this showing, as being anything attached to, or dependent on, the site as a place *per se*.

The statements establishing these facts are to be found in the official Reports of the Superintendent of Prisons, Selangor, from 1891 onwards.

They show that prisoners were almost continuously in residence on this Pudoah site from the beginning of 1891. The Superintendent of Prisons reports for that year: 'The building of a new gaol was commenced in January, on a site admirably adapted for the purpose, situated on the Pudoah road. Temporary wards are being built for the prisoners, in order that they may be removed at once, and employed clearing the ground for permanent buildings.'

In 1892 he reports: '*New Gaol*.—On December 12 last (1891) thirty prisoners were located there, and fifty the following night. At the close of the year sixty were there. . . . All food is taken from the Old Gaol, and any prisoners complaining sick are sent back there under escort.' In 1893 'an average of 117 were employed in building the New Gaol. . . .' 'The management of the gaol has been difficult owing to the number of prisoners located at the New Gaol necessitating the supervision of two separate establishments.'

In 1894 '129 prisoners were employed daily [at the New Gaol] on an average.'

The same reports show that the average daily strength of prisoners for the whole prison was as follows:

1892	177	1895	390
1893	283	1896	372
1894	285					

When Travers states, as he does in his memorandum, therefore, that 'the Pudoah Gaol was *occupied in January, 1895*, previous to which *all* prisoners were confined in the Old Gaol,' it is clear that this statement is vitiated by a serious omission. It is, as an account of facts bearing upon the epidemic, so far from complete, that it omits to say that some 50 per cent. (or more) of the prisoners had been constantly residing at the New Pudoah Gaol site all the time of its building for the four and a half years preceding the outbreak. When it is said, 'The outbreak occurred *immediately after a change in the location* of the prisoners,' this, too, is a statement incompletely representing the facts, since the first cases happened, by Travers's own showing, in August, 1895, whereas the final official transfer to the New Gaol had been made on January 1—seven months earlier.

But if a wholesale and abrupt change of location of the prisoners, such as that invoked to explain the 'outbreak,' never really took place,

the remark will cause the less surprise that neither did any such outbreak, confined to and caused by influences limited to the gaol, ever occur for which any such unusual explanation need have been sought.¹

Travers, in his first memorandum, gives tables in which he states that the 'number of cases of beri-beri admitted to the infirmary' in the Pudoh Prison numbered in 1895, 158; in 1896, 478; in 1897, 275 cases; and adds the remark as to *all* of these cases that they were '*contracted in gaol.*'

In his 'Further Observations' (1902), in a table in which he analyzes all the cases (386 in number) which were treated in the gaol during the period of experiment, he again describes them *all* as having been attacked (whether for the first or second time) *within the gaol itself*.

In no part of either memorandum is it so much as hinted that any of the prisoners *entered gaol with the disorder* already fully developed. Yet in 1895 a quarter, in 1896 a sixth of all the cases of beri-beri treated in the gaol *came in with it*.

H. Wright quotes Travers as saying: 'During the first half of the year 1895, 17 prisoners were admitted to the New Gaol suffering from acute beri-beri, but no cases originated in the prison itself' ('Study,' para. 554, p. 38).

In 1895 McClosky, quoted in his annual report by Travers, states: 'Beri-beri attacked a large number of prisoners during the year—152 in all—with 31 deaths, as compared with 4 cases and no deaths in 1894.'

In 1896 McClosky reports: 'The number of admissions for beri-beri was 499 . . . the majority of the cases were recurrent. . . . Only 74 short-sentenced prisoners were attacked out of 499 cases.' But, from a table compiled by McClosky, given in the 'Study' (p. 39), it appears that 24 more cases were admitted to the gaol with the disorder during the latter half of 1895. Of the whole number of cases treated at the prison in 1895 (152), it appears that 41 had the disease upon committal, 33 were relapses or recurrences; only 78 were developed in the gaol.

Next, a collation of McClosky's table with the figures furnished by Travers in his second memorandum shows, that of the 499 cases of 1896, 264 were 'new cases,' supposed to be contracted in the gaol; 155 were described as relapses; no less than 80 were brought into the gaol with the disorder already developed.

In 1897 McClosky says: 'Out of 297 cases of beri-beri treated in the gaol only 97 were new cases who developed the disease in gaol, the remaining 200 cases were chiefly made up of repeated relapses. This tends to show that the poison is becoming attenuated. Out of 276 cases admitted during the year, only 52 were in short-sentence prisoners.'

¹ The facts and figures next cited are taken, as before, from the annual reports to Government of the Superintendent of Prisons, from medical reports made to Government by Dr. Travers himself, from a Second Memorandum of his, presented to Government, entitled 'Further Observations on the Rice Theory of Beri-beri, being a reply to Dr. Braddon's Criticisms,' etc., from a table compiled by Dr. McClosky, the medical officer in charge of the gaol during the epidemic, published in his 'Study' (No. 1 of vol. ii. of the Institute of Medical Research) by Dr. H. Wright, and from sundry facts reported by the latter observer in the same pamphlet.

488 THE CAUSE AND PREVENTION OF BERI-BERI

There are no returns to show how many of the 97 which were not relapses entered gaol with the disorder. But it is to be supposed, in spite of McClosky's saying the 97 were 'new' cases, that some at least of them must in this, as in other years, have entered the gaol with beri-beri.

In 1898 there were only 73 cases of beri-beri treated in the gaol, with 2 deaths. Of these it appears 20 were relapses, 16 were imported, 20 were new cases, the remainder being cases left over from the preceding year.

I put in the form of a table the number of cases occurring at Pudoh, 'contracted in gaol,' according to Travers' first published memorandum, the different account of the same cases given by himself and McClosky in other reports, the number of cases which entered gaol with the disorder, and the relapses or recurrences in the three years 1895-1897:

Number of Beri-beri Cases treated in the Pudoh Prison, Selangor.

Year.	New Cases 'contracted in Gaol,' according to Travers' First Memorandum. ¹	(a) New Cases 'contracted in Gaol,' according to Travers' Annual Reports, McClosky, and H. Wright.	(b) Cases admitted to Gaol with Beri-beri.	(c) Relapses or Recurrences.	Total of (a), (b), (c).
1895	158	78	41	33	152
1896	478	264	80	155	499
1897	275	97	?	200	297

The astonishing discrepancy between the first and second columns in this return is explained by the third and fourth, the important facts revealed by which put an entirely new complexion on the state of affairs prevailing at Pudoh.

Instead of the picture painted by Travers of an enormous number of cases of beri-beri (911 in three years) developing suddenly at a place, and in a community previously entirely free, we have, then, the representation of the occurrence of a large number of cases it is true, but a great proportion of them were relapses from primary attacks, while of the latter not less than a third certainly, and many more probably, were imported.

It appears that, while in the three years preceding 1895, when three-fifths of the prisoners resided at the Old and two-fifths of them at the New Gaol site, only 9² cases of beri-beri were imported into prison: in 1895, 41, and in 1896 no less than 81 brought the disease with them, most of them having it, McClosky says, in acute stages on admission.

This, at once the most surprising and most significant of the facts connected with the outbreak at Pudoh, was one which should have made it manifest that the gaol, instead of being merely the seat

¹ Published in *Journal of Tropical Medicine*, August 1, 1902.

² Travers' first statement; fourteen or eighteen in later tables.

and 'focus' of an original outbreak of beri-beri, was but sharing in a wider epidemic.

If the number of cases developed in the New Gaol after August, 1895, was extraordinary, as compared with the freedom of the Old Gaol from them before that date, not less so was the abnormal proportion of persons already having the disease—'most of them in acute stages'—on admission to the prison. Whereas in 1893 and 1894 there were only 8 such cases; in 1895 and 1896 there were no less than 121.

Of the one phenomenon, as of the other, the simple explanation is to be found in the general increased prevalence of the disease which has been shown to have occurred at or about the period 1894 to 1896, not only throughout the other native States and the Colony, but in parts of the world entirely unconnected with them, and as remote as Burmah, Ireland, France, Germany, America, Australia, and Fiji.

In the native States the fluctuations in the incidence of beri-beri from year to year have been shown to be identical, not, of course, in extent, but in direction at almost all stations, and to this rule the epidemic at Pudooh after 1895 formed no exception.

How close this correspondence was may be seen in the table, in which the prevalence and case-mortality at Pudooh are compared with several other stations.

Case-incidence of Beri-beri per 1,000 at Pudooh Gaol and some other Stations, 1894-1902.¹

Institution.	1894.	1895.	1896.	1897.	1898.	1899.	1900.	1901.	1902.
Kuala Lumpur Old Gaol	7	—	—	—	—	—	—	—	—
Pudooh Gaol	—	140	486	433	139	159	332	350	770
District Hospital, Kuala Lumpur ..	324	305	391	414	274	302	245	185	239
All out-station Hospitals, Selangor ..	371	399	415	374	205	100	235	166	160
Batu Gajah Gaol ..	73	71	137	124	135	151	214	268	129

Scrutiny of the figures shows (what is more apparent on the charts already given in the text) that between 1895 and 1901 every variation in the incidence of beri-beri in the prison accompanied a similar, though less abrupt, change in its prevalence outside, both in the same area as that in which the gaol was situated—the Kuala Lumpur district—and in the country outside that area.

The same feature characterized the mortality returns. With every rise

¹ In the gaols the ratio taken is the proportion of cases of beri-beri per 1,000 sick admitted to infirmary of all nationalities, since in the gaols all races are fed on the same rice, and all suffer equally from beri-beri. In outside institutions the same ratio is taken applied to Chinese only, since, as has been shown in the text, the numbers of other nationalities affected form an insignificant proportion ($1\frac{1}{2}$ per cent.) only of all the cases.

490 THE CAUSE AND PREVENTION OF BERI-BERI

or fall in the case-mortality from beri-beri at the gaol there occurred a similar change in the rates outside.

Case-mortality per 1,000 treated, in various Hospitals, 1894-1902.

Institution.	1894.	1895.	1896.	1897.	1898.	1899.	1900.	1901.	1902.
Pudoh Gaol Infirmaries	—	203	94	185	29	86	34	23	18
District Hospital, Kuala Lumpur ..	289	227	185	231	157	208	209	216	188
District Hospitals, out-stations	173	147	197	224	130	152	125	101	158

Only in the last two years in the incidence-table and the last one (1902) in the mortality-table does this correspondence fail.

The divergence in the extent of incidence in the gaol in the latter year may be explicable on the ground that more critical examination of convicts led to many being included in the returns of a degree of mildness which would not be represented among hospital inmates outside, and may even have failed to be included among the prison returns of earlier years.

Reference to the tables of statistics and the charts attached to the body of this paper will show that the wave of increase which affected Pudoh in 1895 was marked also at thirty out of the thirty-two other hospitals open at that date, of which I have obtained statistics. In the case of twenty-five of them the increase continued on into 1896, and in fifteen into 1897. The decline following (as that preceding) was equally widely shared, being marked at thirty-five of thirty-seven hospitals open in 1898, two remaining level.

The fact that there was a great exacerbation in the factor which determines beri-beri just at the juncture when the removal of the prisoners from the Old to the New Gaol was accomplished is thus made plain. Pudoh was not singular, even among gaols, in experiencing its effects. The increase justifies the belief that had there been no change effected in the locality of the prisoners at that time the epidemic would have occurred just the same at the Old Gaol, an inference which is strengthened almost into certainty by the fact that beri-beri did occur among the lunatics who occupied the Old Gaol when the prisoners had vacated it.

While the general epidemic wave of increase sufficiently explains both the unusual number of cases of beri-beri entering the gaol, and the occurrence of the disease after admission among those admitted free of it, there were other causes at work to make the incidence upon inmates of Pudoh more marked than elsewhere, and more severe probably than it would have been without them.

One of these factors (which has been already touched upon) was that in prison, cases evincing only the slightest manifestations of the malady become recognised, which would pass unnoticed outside, and during the

earlier stages of an epidemic probably in gaol also. These are included in the statistics, and exaggerate the apparent incidence of the disease in prison, as compared with hospitals outside (in which chiefly severe cases are treated), and of the later years of an epidemic as compared with the earlier, in the prison itself.

Another event which affected the condition of the gaol and, at least in part, accounted for the much larger number of convicts coming into prison with the disorder, which happened in 1895, and has been left unnoticed, was that after this date the convicts from all parts of the State were sent in there, whereas in the Old Gaol only those drawn from the Kuala Lumpur area, with a few long sentences from the districts, were accommodated.

The Kuala Lumpur district having been for some years freer of beri-beri than the out-districts, the effect of this change would be to increase the number of importations with severe beri-beri, and probably of those most likely to get relapses also. The condition of the New Gaol, therefore, after 1895 could properly have been compared, not with the Old Gaol alone before that time, but with all the gaols of the State taken together.

These are statistical matters not greatly, perhaps, affecting the main conclusions to be drawn as to the causes of beri-beri at Pudo. But there were other two which it can hardly be doubted greatly helped to intensify the epidemic. One of these was a change in the condition (as compared with previous years) of the mass of the individuals actually admitted.

Although beri-beri is known to find its most numerous victims among the well nourished (healthy young male adults), there is no reason to suppose that the weakly or indigent are made through these conditions less liable to get it, and the presumption must be that they are more likely to do so when the influence acts upon them.

These two apparently opposed conclusions are reconcilable on the supposition, that the healthy acquire beri-beri more readily, merely because they are more fully exposed to its cause than are the weakly and sick—a state of affairs easy to understand when the cause of the disease is conceived to lie in food; since of this the healthy may eat much, while the sick and indigent, by the necessity of their case, get but little. But when, through change of circumstances, individuals of the latter class should become heavily dieted with a poisonous rice, it seems reasonable to suppose that they should succumb to its effects more rapidly than the healthy.

The prison had always received a number of vagrants, but the records show that in 1895 the proportion of these had assumed unusual dimensions. In the Prison's Report for that year the complaint of the gaoler is noted that of 3,120 committals no fewer than 747 were vagrants. In 1896 they numbered 662, and it must be supposed that they largely helped to swell the list of patients with beri-beri.

Another condition which predisposes to beri-beri, more, indeed, than any other, is the fact of having already had it. In Pudo, at all events, the relapses outnumbered the original cases.

Now, records again show that at Pudo a very large number—at least

50 per cent.—of the committals in each year are recidivists, one half of whom, again, have been convicted twice, and the other half thrice before. Since one attack predisposes to, rather than protects against a second, the continual reflux to gaol of prisoners, many of whom had already had it, must gradually result in the establishment of a prison population peculiarly prone to beri-beri.

Nothing is said in the reports as to how many of the beri-beri patients were recidivists.

Last, there is the circumstance, not mentioned in his account of the epidemic by Travers, which, in the light of the evidence connecting rice with beri-beri, cannot but be regarded as having had more to do with the increased incidence of it at Pudooh than anything else, that in the year of the 'outbreak' the quantity of rice entering into the dietary of the prisoners had been largely increased.

Prior to 1895 the ordinary diet (given to all prisoners, except those doing three months and under, thrice a week for the first six, and daily after the first six months of their sentence) had contained: Rice, 14 ounces, (with 4 ounces of fresh fish, 5 ounces of beans, and 1 ounce of cocoanut-oil, in addition to meat, vegetables, and flour). But in 1895 a new scale was adopted in which the rice was increased to 19 ounces, the fresh fish replaced by salted, while the beans (of great proteid value) were omitted. The other articles, and the penal diet (given to all prisoners serving under three months daily, and to others alternately with ordinary diet for six months), remained the same.

The amount of rice consumed daily by all prisoners serving six months or more was thus increased considerably, while the quantity of nitrogenous elements was cut down.

The increase in the rice would be 15 per cent. daily for those serving under six months, and 30 per cent. daily for those serving more—the long-sentenced prisoners.

Now, it is precisely this class who, experience shows, are almost alone liable to contract beri-beri, the difference between them and the short-sentences in Pudooh itself, in this respect, having been shown to be as 400 to 1. Supposing rice to be the vehicle through which the poison causing beri-beri is conveyed, it is obvious that the increased quantity of it consumed in the new diet might be quite sufficient to cause the disease in those who on the old diet would have remained unaffected.

Especially would this be the case when the rice in general use might be supposed to be more toxic than ordinarily, and it has been sufficiently demonstrated that there was a definite general exacerbation in whatever may be the cause of beri-beri at this time.

The facts considered, revealing as they do a quite different state of affairs prevailing among Selangor prisoners before, and after the occupation of the New Gaol, might alone be held sufficient to determine the outbreak of a malady, the cause of which was already abroad in an acuter form than usual, especially if that cause were rice.

They explain why, the disease prevailing, Pudooh should have been particularly attacked, and placing the epidemic there in its true light as one among many due to a common agency, they confirm the con-

clusion earlier arrived at, that the essential cause, at Pudooh as elsewhere, lay not in the place as such, nor in anything peculiar to and generated at the place, but in other conditions.

For those who believe in the dependence of beri-beri upon a living infection, some germ properly or facultatively parasitic in man, the introduction of so many fresh cases of the disease into the New Gaol from outside will at once explain the spread and continuance of the disease within its walls. To such it will be vain to point out that cases were constantly introduced for many years into the Old Gaol (as in many other such institutions) without any epidemic resulting; that in the New Gaol, where every prisoner had a separate cell to himself, so that there was no overcrowding, the hygienic conditions were much more favourable than at the Old Gaol, where they lived together in association-wards; that a brick-and-mortar cell, daily washed out by its occupant, who has little more to do than keep it clean, is the least likely nidus upon which germs should grow.

On the other hand it is to be observed that on an infection theory it is easy to show that there was, or may have been, no epidemic at Pudooh at all—none, that is, composed of cases originated in the place. One has only to postulate a sufficiently long incubation period—and, whatever the cause, it is certain that the time required to produce the disease is a long one—and every case may be explained as the result of *infection before entering* the prison.

But the experiment at the two gaols itself affords the most convincing evidence against the possibility of beri-beri being caused by any form of infection transportable by individuals.

Thus, if beri-beri is due to a specific germ which produces its effects by multiplying in the body, it can make no difference to the progress of the malady (provided that it continues to be properly nourished) *where* the body is placed. It can certainly in no way affect the progress of the disease to maturity in an infected individual, whether he is housed in this or that locality, this or that prison.

This, which (as exemplified in the recovery of patients upon removal from the circumstances under which they became ill) has long been used, and validly, as an argument against the infectious nature of beri-beri, applies not only to the later stages, when illness has been produced, but equally to the earlier ones, when, although the assumed infection has occurred, the symptoms entailed by it have not begun.

Now, at Pudooh Gaol it is a fact that numerous persons entered in all stages of the disorder. There was, therefore, some constant source of the assumed infection without; and it follows that, whatever the period of incubation taken, some of those admitted to the prison must have been, though apparently healthy, in reality already in the incubation stage of the disease. In such persons it is obvious that the symptoms must have appeared after admission, to whichever of the two gaols they should have been assigned.

However the prisoners may have been divided for the purpose of observation between the Old and New Gaols (provided that the number

kept at either were sufficient to afford a legitimate basis for conclusions), it must have happened that some of such 'larval' cases should have matured, have fully developed the disease at each prison—at the Old Gaol, therefore, as well as the New.

But this did not happen.

Although two-fifths of all the prisoners, forming a daily average strength of over 100 persons, were kept under observation at the Old Gaol for the nine consecutive months of the experiment, not a single case of beri-beri originated among them, while at the New Gaol; with a daily average of less than twice the number at the Old, in the same period, 193 men acquired it.

Does not such a result absolutely disprove the possibility of the production of beri-beri through any form of specific infection at Pudooh or elsewhere?

Clearing the ground of this theory, the experiment has the merit of narrowing the etiological issues. The Pudooh results must be reconciled with one or other of the only two remaining theories of origin of beri-beri for which there has ever seemed to be any evidence—those, namely, of intoxication by some emanation produced in the patient's surroundings—a miasm, or 'place-effect,' or, poisoning through food—rice.

The reasons for rejecting the former theory have been already discussed.

They may be summarized as follows:

1. Its inadequacy to explain the selective incidence of beri-beri upon different races, all equally exposed, and under equal conditions in every respect, except food, which actually happens in prisons, lunatic asylums, ships, and even wider regions, of all of which examples have been given.

2. The limitation of the effect (in quoted instances) to members of one religion, or of one social position only, among communities under otherwise identical conditions—except, again, as regards food.

3. The occurrence of outbreaks upon ships and among crews which have never been in contact with or near any beri-beric person or locality in mid-ocean, and many months out of port.

4. The simultaneousness in the rise and fall of the incidence and case-mortality of the disease in places wide apart, and under wholly diverse meteorological conditions.

5. The occurrence of the disease among persons who have no place, no settled dwelling at all, as in the case of Malays, Dayaks, and Dutch soldiers upon 'excursions' and expeditions.

6. The proved uselessness in all cases of measures directed to remove the postulated source of the emanation—disinfection.

The idea of a vapour distilled by whatever agency will not explain these things; but that of a poison conveyed in food will.

It remains to show that, his expressed conviction to the contrary notwithstanding, the results obtained by Travers are, after all, perfectly compatible with the rice theory, as it has been formulated in this work.

It is, fortunately, easy, without impugning any of the asserted

facts, or the good faith with which they have been presented, to determine the error in the original observations, and the fallacy following it which led to the conclusion adverse to rice.

Travers, in his memoranda already cited, and everywhere in his tables and figures in other reports, treats 'prisoners' as if they were all invariably units of equal value.

But this, whether for purposes of clinical observation, or statistical inquiry, above all for the ends of his experiment, they certainly were not.

In the Selangor prisons the vast majority of the prisoners are only committed for quite short terms, most of them for a few days or a month, a small minority for periods exceeding six months, few for more than a year.

At Pudooh the proportion of them sentenced to terms of over six months (long-sentence prisoners) was in 1895 but 5·6 per cent. ; in 1896, 3·8 per cent. ; and in 1897, 4·7 per cent. of the total committals.

Moreover, the committals are not made at once, but are spread all over the year.

The date of committal and term of imprisonment of all the individuals entering the gaol differing, it is obvious that, however groups might be selected originally, their composition must continually vary.

Discharges might be balanced by new admissions, and a fixed or 'average daily strength' maintained in given parties, but, their individual components varying, the actual groups would differ from day to day.

Now, while this might not greatly matter, were the question one of investigating the effect upon individuals of a definite poison, the exact time of application of which should be known, and the results to be expected determinable within a few days : it became of great moment when the object of experiment was an influence, the actual presence of which could only be determined by its effects, and these such as required months perhaps to produce.

If the question be asked, Was there any one definite condition as to which it can be said that it did positively determine the greater or less incidence of beri-beri upon individuals, or classes of them, at Pudooh ? the answer is clear and indisputable—that the length of period of incarceration was undoubtedly such a factor.

Whether because their daily chance of acquiring the disease was proportionately multiplied for every day spent in gaol, or because the malady itself is due to the accumulation of a repeated series of slight effects, or for whatever reason, the fact remains, that the liability to acquire the disease did increase, and varied directly with the length of time served in the prison.

The exact period at which that liability reached a maximum cannot, for want of complete details in published reports, be determined. But the classification of the prisoners into long and short sentences, fortunately, enables us to be certain that it was something more than six months.

During the period of his experiment (October 21, 1895, to July 1, 1896)—I quote Travers' own analysis of the figures—386 cases of beri-beri were treated in Pudooh Gaol.

During the same period over 2,500 short-sentence prisoners were admitted, and among them 170 cases of beri-beri occurred. Including those entering gaol with the disease, relapses, and recurrences, the incidence of beri-beri on short-sentence prisoners was thus 6·8 per cent.

But if primary attacks, incurred within the gaol, only be considered, *the incidence on short-sentence prisoners was but 3 per cent.*

The incidence on long-sentence prisoners during the same period was between 58 and 65 per cent. !

The number of long-sentenced prisoners remaining in the Old Gaol on December 31, 1894, and transferred to Pudooh on January 1, 1895, was 152.

There were committed during the year 141, and remained at the end of it 153, so that discharges and admissions were nearly equal.

Ninety-nine long-sentence prisoners were admitted during 1896.

The experiment began on October 21, 1895.

The number of long sentences in gaol on October 21, taking the mean for the year, was 152.

To this number have to be added some admitted subsequently—viz., during the last two months and ten days of 1895. Since, by the conditions, those who should spend less than six months in gaol were little likely to acquire beri-beri, all the long sentences admitted after January 1, 1896, would have to be excluded as failing to fill this condition; but, in order that the issue may not be made to appear to depend on too narrow a basis, only the admissions subsequent to March 1, 1896, will be omitted on this ground.

The additional long sentences admitted between October 21 and December 31, 1895, would be one-sixth of the total returns for the year (141), or 24 persons. Those for January and February, 1896, would number one-sixth of the total for that year—199—or about 16. This gives the total possible subjects of beri-beri to be acquired entirely in gaol during the experimental time as 196—say 200. (If a six-months' period of incubation be adhered to, the number of probable subjects would be less—176.)

Among this lot of 200 possible subjects during the nine months of experiment, 217 cases of beri-beri were treated. Five entered gaol with the disease, 117 individuals acquired it in gaol, and 95 had relapses.

The incidence for primary attacks was thus between 58 (at a minimum), and 65 (for a maximum) per cent. Counting all attacks, it was more than cent. per cent.

In 1896 there were but 74 cases of beri-beri of all kinds among 2,459 short-sentence prisoners admitted—a ratio of 29 *per* 1,000.

The incidence on long-sentence prisoners during the same year (primary attacks only) was between 61 *and* 66 *per* cent.

One hundred and fifty-two long sentences remained on December 31, 1895; 99 were admitted during 1896. The total passing through, who could have acquired beri-beri, after a four-months' incubation

only, was therefore 218 (or after a six months' incubation, 201); the new cases furnished by them were 103, a ratio of between 472 and 524 per 1,000.

In calculating the number of possible subjects of primary attacks of beri-beri in 1896, some 50 or 60 have to be deducted as having already incurred the disease in 1895. Deducting 50 on this count, the rates of incidence become 613, or 666 per 1,000.

The relapses in this year among long-sentence prisoners were 161, so that the total attacks on them were more than 200 per cent. !

In 1897 only 52 cases of all kinds were recorded among 2,544 short-sentence prisoners committed—an incidence again of 2.9 per cent.

The incidence upon long-sentence prisoners was between 40 and 55 per cent.

One hundred and twenty-six long sentences were committed, of whom 84 would thus be possible subjects at a four, and 63 at a six months' incubation period.

I have not the record of the number remaining at the end of the year, but, seeing that no less than 103 of them got beri-beri in 1896, there would have been but few left who were capable of incurring first attacks in 1897. Probably not more than a score of individual convicts can have been so circumstanced; so that the possible subjects in 1897 would number either 106 or 83. Among them 48 new cases occurred, giving a rate of between 424 and 554 per 1,000.

The number of actual relapses in this year was 200 !

In short, while during the years discussed the proportion of short to long sentence prisoners incarcerated was as 20 : 1, the liability of the two classes to get beri-beri was as 1 : 20. In other words, the long sentences proved 400 times more liable to get it than did the others.

This enormous disparity in the liability of the two classes of prisoners to acquire the disease makes it obvious :

1. That no group of them to be put under observation for the purpose of testing the presence of the causal agent of beri-beri can be regarded as satisfactory which should be wholly or even principally composed of short-sentence prisoners, since not more than 3 per cent. of these could be expected in any case to get the malady.

2. That the absolute number of persons put under observation would have to be very large—at least, several thousands—to exclude the probable error of ratios in dealing with small quantities.

It follows that no experiment designed to compare the prevalence of the disease by exposing parties of prisoners at each of the two Selangor gaols could afford ground for any valid conclusions, unless care were taken to secure that there should be included in each party a sufficient number of individuals, each of whom should remain continuously under observation for a term at least as long as that which is necessary to acquire the disease.

The same relative proportions of all classes of prisoners should have been maintained in the parties kept at each place. An exact record

should have been kept of the date at which each prisoner was admitted to, and the period for which he was kept at, either prison.

There is nothing in the account of his experiment by Travers to show that these conditions, absolutely essential to the attainment of correct results, were in any way either realized or observed.

His treatment, in more than one set of statistics, of all 'prisoners' as units of identically equal value, must be taken as showing that he actually regarded them as such at the date of the experiment. It had evidently not then become apparent to how great an extent the liability to beri-beri increases with length of exposure to its cause, so emphatically shown by the great difference in its incidence upon the long and short sentences at Pudo. The question of a possibly lengthy incubation period seems to have been wholly ignored.

In the absence of evidence that these conditions were appreciated and enforced in his experiment at the two gaols, it is impossible to attach to the inference which Travers drew from it the value it might otherwise have had.

For the establishment of a conclusion so important as that which is claimed as the result of the Pudo experiment—namely, that epidemics of beri-beri may occur, in the causation of which food (rice) plays absolutely no part—something more critical is required than mere summaries and bare generalizations.

At least, a detailed record of the individuals experimented upon, their condition, and the term which they spent under observation, ought to have been accessible. But the experiment seems not to have had in its author's eyes at the time that importance which has, unfortunately, become attached to it through publication later, and no such record of the actual details was kept, or is now available.

The writer at once¹ pointed out the objections to Travers' conclusion which the sources of error now alluded to naturally formed, and showed how it was more than probable that the reason why none of the prisoners exposed at the Old Gaol got beri-beri was because none of them were kept there long enough to do so.

Travers, replying to this criticism, was unable to produce any detailed record of the individual prisoners sent to the two gaols, or of the terms they individually spent in them. But he said:²

'The prisoners sent to the Old Gaol were passed by me personally. No selection of any kind was made except that each man was examined for symptoms of beri-beri. Long-sentence prisoners in chains (including one now in the Pudo Gaol undergoing sentence for life) and short-sentence prisoners were sent indiscriminately, in order that a fair sample of the prisoners in the Pudo Gaol might be experimented on. The numbers were kept up by the transfer of batches of healthy prisoners

¹ 'The Light of Local Experience on the Rice Theory of Beri-beri: Where it Fails.' Perak Government Printing Press, 1902.

² 'Further Observations on the Rice Theory of Beri-Beri: Being a Reply to Dr. Bradden's Criticisms of certain Pudo Gaol Experiments,' by E. A. O. Travers, p. 3. Selangor, 1892.

sent from the Pudooh Gaol to replace those discharged from time to time on expiration of sentence.'

Such a statement, made from recollection merely, of facts which occurred seven years before, and unsupported by any kind of record made at the time, can hardly be held to dispose of the grave objections to the conclusion drawn from the experiment at the two gaols.

It does not state *how many* long sentences were sent to the Old Gaol, and the partial representation of facts already noticed in some of the statements of the 'Memorandum' makes it quite possible that, although the plural is used, the actual number sent may have been no more than two or three. The express mention that 'no selection of any kind was made' permits the inference that special care to secure a proper proportion of long sentences in the Old Gaol group was not made either.

The objection taken here is not captious. It will appear immediately that it is well grounded in fact.

Fortunately, in the very paper just quoted, although no complete record of all, even the principal, details of the experiment is given (or, indeed, possible, none having ever been kept), such evidence as to the distribution of the long-sentence prisoners in the two gaols is furnished by Travers as places the true explanation of the absence of beri-beri from one of the gaols during the experiment beyond any serious doubt.

It has been shown that the total number of long-sentence prisoners passing through the gaol during the eight months and nine days of experiment (October 21, 1895, to June 30, 1896) could not have exceeded 234. Among these as much as six months of their term (the demonstrated average period of latency of beri-beri at that gaol and that season) could have been spent within the period of experiment by not more than 180 individuals.

Between August 1, 1895, and June 30, 1896, Travers states¹ 279 *individual prisoners* acquired beri-beri for the first time in Pudooh Gaol, of whom 122 were long and 157 short sentences. Fifty-eight of these same cases, I find from McClosky's table,² happened *before October 21*, the date of beginning the 'experiment.' Divided in the same proportion as obtains with all the prisoners, 33 of them would be short and 25 long sentences. The latter have to be deducted accordingly from the tale of 122 long sentences who got beri-beri during the period of experiment, leaving $(122 - 25) = 97$. And since those who got it—had already had it—before October 21 cannot very well be included among possible subjects of primary attacks after October 21, the same 25 must also be deducted from the number of the latter, leaving $(180 - 25) = 155$.

We have, then, 155 as the number of individual long-sentence prisoners who could have spent the six months' term necessary to acquire beri-beri in the gaol during the experimental interval, and 97 as the actual number of individuals who did acquire it.

In the experiment about three-fifths of the 'average daily strength' of prisoners were kept at Pudooh Gaol; two-fifths were sent to the Old Gaol.

Now, had all the 155 long-sentence prisoners present in gaol on October 21, or admitted subsequently, been divided (as they should have

¹ Annual Medical Report of Selangor, 1902.

² *Loc. cit.*

been) in the same proportion, three-fifths of them, or 93 individuals, would have been kept at Pudoh, two-fifths, or 62 individuals, sent to the Old Gaol.

But all the 122 long-sentence prisoners who acquired beri-beri, including the 97 affected during the experimental period, did so at the Pudoh (New) Gaol, proof enough that they, at least, had not been sent to the Old Gaol.

Next, the certainty that 97 of the possible 155 patients were kept at Pudoh entails this further consideration. It is unlikely in the extreme that *every one* of the long sentences who were kept at Pudoh should have become attacked. And, as a matter of fact, this did not happen. It has already been shown that the case-incidence on prisoners of this class during the period was 65 per cent. For every two individual long sentences who got the disease there, it is clear, therefore, that there must have been one in addition who escaped. Those who actually got it numbering 97, the *total of those exposed must have been 145*. At least 145, therefore, out of the 155 possible patients were kept at Pudoh, leaving *ten only who could have been sent to the Old Gaol*.

Without attaching too much importance to deductions, such as drawn from these statistics, which, after all, are incomplete, insistence may be laid upon the facts, which are clear and certain, that in 1895 only 141, and 1896 only 99, long-sentence prisoners entered Pudoh Gaol; that between August 1 in the former and June 30 in the latter year no less than 122 of these individuals got beri-beri at Pudoh.

Who can believe, in face of this last indisputable fact (the authority for which is Travers' own statement), that anything but the most exiguous number of long sentences could have been sent to the Old Gaol? The proportion sent there—Travers having stated that there were certainly some—must have been very small, far too few to afford a proper basis for experiment, certainly not enough to exclude the probable error in dealing with small ratios. Moreover, seeing that as many as one-third or more of this class constantly escaped the disease during any given year, even at Pudoh, the half-dozen or so of individuals sent to the Old Gaol may very well have been all of them in this category.¹

Finally, it is to be observed, *in regard even to the few long-sentence individuals stated to have been sent to the Old Gaol, there is no assertion, still less any proof, that any of them were kept there for the period (either four or six months) necessary to acquire the disease.*

The fact that the prisoners kept at the Old Gaol did not get beri-beri in this experiment is explained, therefore, simply enough by the conditions. The group sent to the Old Gaol became unintentionally a selected one—one of which the component individuals were in residence too short a time to be capable of getting the malady. There can be little doubt that, had a sufficient number of long-sentence prisoners been

¹ It is possible that, since all prisoners who got beri-beri at Pudoh from October, 1895, to March, 1896, were sent to the Old Gaol for treatment, they may have accounted for Travers' recollection of long-sentence prisoners being present at the Old Gaol.

sent to the Old Gaol, and kept there for as long a time as they were in residence at Pudoh, beri-beri would have prevailed among them. The proof of this is that when, after the experiment on prisoners, other individuals were incarcerated at the same site and in identically the same buildings, but as inmates of a lunatic asylum, for the greater length of time for which the inmates of such institutions naturally are incarcerated, beri-beri did occur, even severely, among them, and has been prevalent there ever since.

To sum up: meritorious though its conception was, the Pudoh experiment, as affording *evidence* against rice as a cause of beri-beri, must be held to be inconclusive. The conviction to the contrary expressed by its author is the result of a partial statement of facts only partially considered.

One of many such generalizations based on an examination of insufficient evidence, it affords no ground for opposing the theory that beri-beri is incurred through the consumption of certain sorts of rice.

DIETS AND BERI-BERI IN THE SINGAPORE AND SELANGOR GAOLS.

1. In the *Singapore Criminal Prison* for many years—1869 to 1875—the dietary for native prisoners comprised little else than rice, and of that an excessive quantity.

The scale was as follows:

Rice	28 ounces daily for all.
Vegetables	6 " " "
Salt	$\frac{1}{2}$ ounce " "
Curry stuff	$\frac{1}{2}$ " " "
Lard	$\frac{1}{2}$ " for Chinese, cocoa-nut oil for Malays and Tamils.
Fish (fresh)	4 ounces thrice weekly for Chinese, four times for Malays and Tamils.
Fish (salt)	$2\frac{1}{2}$ ounces thrice weekly for all.
Meat (fresh fat pork)	4 " once a week for Chinese only.

This 'rigorous' diet was not given every day. Ten days in each month every prisoner had 'penal' diet. It consisted of rice, 28 ounces; salt, $\frac{1}{2}$ ounce.

Allowing for this substitution, and the variation of the articles on different days, the ultimate daily average ration became:

	Rice.	Meat.	Fish (Fresh).	Fish (Salt).	Fat.	Vegetable and Adjuncts.	Proportion of Rice to Whole Rations.
	ozs.	oz.	ozs.	oz.	oz.	ozs.	
For Chinese ..	28	0.38	1.14	0.11	0.34	5	} 79 per cent.
For Malays and Tamils ..	28	—	1.52	0.71	0.34	5	

502 THE CAUSE AND PREVENTION OF BERI-BERI

While this dietary was in force it is not surprising to learn the prisoners were constantly very sickly, and the mortality was great. Rowell gives the following figures :

Year.	Average Daily Strength of Prisoners.	Actual Deaths.	Rate of Mortality per 1,000 of Strength.
1869	2,055	55	26
1870	1,981	48	24
1871	1,933	53	27
1872	1,927	52	27
1873	1,986	48	24
1874	763	34	24

Considering that the majority of the committals to the prison were able-bodied young adults, and that less than one-half of them served as much as six months in the gaol, the mortality revealed by these figures is very great.

Although there was no actual record of beri-beri being present in the gaol during these years, yet there can be little doubt of it, since the disease was very prevalent without the prison walls, and in the Report of a Committee of Inquiry held in 1880, the medical officer is stated to have said : ' Although there is no reliable evidence that beri-beri existed in the prison before 1875, yet there is presumptive evidence that this was so, the cases having been recorded under the names of " general dropsy," " chronic rheumatism," and " paralysis." ' ¹

However this may have been, beri-beri broke out in April, 1875, and was soon recognised to be severely epidemic. From that date onward it was seldom absent from the gaol until 1885, when, coincidentally with a great reduction in the amount of rice given, and the improvement of the diet in other particulars, it totally disappeared from among the prisoners, though continuing with unabated activity among similar classes outside the prison.

This exemption of the prisoners continued for twelve years, but in 1897 (the proportion of rice in the dietary having again been increased) the disease reappeared, and continued to scourge the prison severely till 1903, in which last year there was a general and simultaneous diminution of the disease in almost every quarter where it had previously prevailed.

The history of the fluctuations of beri-beri in this gaol is thus a record of nothing more than alterations in diet, of the dependence of the disease upon the rice-element in which it, indeed, affords a most instructive example.

The following account of these changes in the various years is gathered from the Government records :

¹ *Straits Settlements Government Gazette* (Blue-Book, Straits Settlements), 1881.

1875.—In this year the state of affairs was :

Average strength of prisoners..	667 ²
Cases of beri-beri	615
Deaths from beri-beri	69

The case-incidence per 1,000 of strength was, therefore, 921, and the mortality from beri-beri per 1,000 of prison-strength 103. In addition, the general sickness-rate was increased, the deaths from all other causes amounting to 21 per 1,000.

1876.—‘ In January, 1876, the “ penal ” diet of rice and salt for ten days in each month was discontinued.’¹

The effect of this alteration was to raise the amount of proteid food (meat and fish—fresh or salt) in the *daily* ration from 2½ to 3½ ounces, and to reduce the rice-component to 71 per cent. ‘ In July a more substantial general diet was introduced,’² details of which are not given,³ but Rowell says of it later : ‘ The first beneficial change was made, I believe, at Major Grey’s (the Prison Governor’s) suggestion in August, 1876, and consisted in slightly increasing the proportion of animal food.’ ‘ During the year prior to that in which it was introduced 615 cases of beri-beri occurred, among which there were 69 deaths, while during the same period in the following year there were only 108 cases with 8 deaths.’ Though not certain, from this statement it would seem that all these cases and deaths took place in 1876, since the Prison Report for the next year (1877) says the ‘ health of the prisoners was good, the sick not amounting to 5 per cent.’⁴

The conditions in 1876, therefore, according to this statement, were :

Average strength	667
Cases of beri-beri	108
Deaths from beri-beri	8

or a case-incidence of beri-beri of 159 per 1,000 strength, and a mortality from it of 12 per 1,000.

But a table prepared by Rowell, published by the Committee of Inquiry in 1880,⁵ shows that 54 deaths from all causes happened in 1876—81 per 1,000 strength. It must be supposed that a large proportion of these, too, were really beri-beri, since during the six years preceding 1875 the mortality from all causes, heavy enough, only equalled 24 per 1,000, and this was supposed to have included beri-beri. Allowing the same rate—24 deaths per 1,000—to cover all causes exclusive of beri-beri during 1876, we should have a balance of 57 deaths per 1,000, which should be the result of beri-beri only. In the chart given in the text I have used the official return of 12 per

¹ Straits Settlements Blue-Book, 1877 : Annual Report on Prisons.

² *Ibid.*

³ *Ibid.*, 1881 : Annual Report Medical Department, p. 36, paras. 56, 57.

⁴ *Ibid.*, 1878 : Report on Prisons. ⁵ *Ibid.*, 1881, p. 97.

504 THE CAUSE AND PREVENTION OF BERI-BERI

1,000, but it must be held to be given subject to the correcting inferences now made.

1877.—The average strength was 823; there were only 22 deaths from all causes—26 per 1,000 of strength. There were but two cases of beri-beri recorded.

1878-1881.—In these years the disease became more prevalent than ever, as the following table show :

Year.	Strength.	Cases admitted.	Deaths.	Incidence per 1,000.	Mortality per 1,000.
1878	923	142	23	153	67
1879	826	316	66	388	128
1880	789	588	63	748	79
1881	688	229	23	332	29
1882	688·2	87	23	126	33
1883		19	nil	27	nil
1884		7	nil	10	nil
1885		55	4	98	7

In the Administration Reports for Singapore but few details of the medical history of the gaols is given, but all these institutions were continuously under a uniform régime, so that any recorded alteration of diet at one gaol applies equally to all. Nothing is said of any change at Singapore to which the recurrence of the disease there might be attributed. But in the Report of the Penang Prison¹ for 1878 it is stated that 'a new reduced scale of diet was commenced in April, 1878; the health of the prisoners did not appear to suffer from the reduction.' This reduction affected evidently not the rice, but the proteid and other adjuncts, for I find from Bentley,² who quotes Rowell's Report of June 14, 1881, that during these years (1878-1880) the rice was as before, 28 ounces daily for short-sentence prisoners, 30 ounces for long sentences. The effect of the 1878 'reduction' was, therefore, to increase the relative proportion of rice in the whole diet.

1879.—In the Prison Report from Penang³ for 1879 it is stated: '*Penal Diet*.—This diet was reintroduced on November 3, 1879, all prisoners except those in the revenue grade being put on penal diet for ten days in each month. Prisoners with sentences of under one month were given it for the first ten days of their sentence.' The effect of the use of penal diet intermittently with the ordinary was to raise the rice-equivalent in the actual daily ration (averaged from both) to 79 instead of 71 per cent.

As the abolition of the penal ration in January, 1876, and the other improvements made in the diet in August, 1876, had been followed by immediate reduction in the beri-beri rate, ending in its almost

¹ Straits Settlement Blue-Book, 1879.

² 'Beri-Beri,' by C. J. Bentley, 1893, p. 11.

³ Straits Settlements Blue-Book, 1880.

complete extinction during 1877, so now the restitution of the diets to their former scale was followed by a renewed outbreak of beri-beri, the extent of which was probably much greater than is revealed by the table given above. The Governor of the prison, in his Report on the year 1879,¹ states: 'The prisoners have been extremely sickly and affected with beri-beri . . . within the first six months, 10 per cent. of the whole number died.' The daily average strength was 754, the deaths were 82. The total of deaths for the twelve months was 105.

1880.—There were yet more cases in the first four months of the year. Three-quarters of the whole prison strength had become attacked—748 per 1,000—and, though the disease was less fatal, something like a panic seized the gaol. Nothing availing—and everything possible was tried, to check the march of the malady—it was decided to evacuate the buildings for a time, and transfer the inmates elsewhere. Accordingly, in April all the long sentences, some 184 in number, were transferred to Penang, and the others were sent, 32 to Malacca, and 150 to the Sepoy lines in Singapore. This, of course, eliminated the disease from the Singapore Gaol, but, what had not been anticipated, and a fact obstinately opposing the view that a local miasm, the 'endemic infecture factor' of the latest exponents of beri-beri epidemiology at Singapore, was its cause (as had previously been insisted upon), the affection followed those prisoners who were sent to Penang, and in that gaol, where no cases (it is stated) had ever previously been known to occur, 63 of the transferred Singapore men were attacked, of whom 4 died. (The 180 survivors were brought back to Singapore on June 26, 1881.) This outbreak at the Penang Prison has long been fallaciously cited in many quarters as a proof or presumption of the transportation of *infection*. This point is dealt with in the text. Its explanation lies in the fact—affording a proof of the lengthy latent period of beri-beri—that it is only, or chiefly, those who stay in prison six or more months, as long sentences do, who are sufficiently exposed to the cause (*e.g.*, long enough under the influence of the toxic rice given them) to get beri-beri.²

The Committee of Inquiry dealing with this outbreak of 1880 in the Singapore Prison reported that 'the prisoners had not been overcrowded in any way. . . . The whole of the prison had been fully repaired, lime-washed, and painted, and fumigations throughout all the dormitories continued to be carried out with regularity.'³

Of the dietary, the same Committee reported that it was 'sufficient in quantity.'⁴ The medical officer (Rowell) expressed his opinion as follows: 'I feel assured that the dietary *per se* cannot be credited with

¹ Straits Settlements Blue-Book, 1880: Prisons Report, p. 1879.

² Rowell expressly declared that there was no evidence of the Penang outbreak being due to contagion, and Bentley ('Beri-Beri,' p. 42) points out that actual cases had been similarly sent from Singapore to Penang in 1875 without any such sequel. This was, of course, to give them the benefit of change of locality.

³ Straits Settlements Blue-Book, 1880: Report of Committee.

⁴ *Ibid.*, Report of Committee.

having been a factor in the causation of the disease. It is a prison diet, but it is one which is liberal, sufficiently nutritious, and varied,' etc.

Nevertheless, it was resolved to try the effect of improving the rations.

In 1881¹ the medical officer of the prison reports: 'The dietary for natives which has been in use for many years has undergone revision, and, concluding that it would be improved by the addition of a larger proportion of nitrogen in its formation, the alteration was submitted to Government and approved. It consists chiefly in the reduction in the quantity of rice, and the substitution of a certain amount of dholl² in its place, with a slight increase in the animal food. . . . The change has been attended with beneficial results, and, though some of the prisoners have complained of its not being enough, there is no doubt that it is ample. . . . I think also that the general health of the prisoners has been improved by it, the disorders of the digestive organs having become less frequent.'

This substitution of 4 ounces of rice by as many of dhal was made in July, 1880,³ and had the effect of reducing the rice-component of the ration once again from 79 to 71 per cent. of the total, as well as of greatly improving its nitrogenous value, so that to the absolute reduction of rice was added a greater relative diminution of its effect. No more cases occurred, it would seem, after this improvement, during that year.

1881.—'The health of the prisoners was good up to November, when during very wet weather beri-beri again appeared. Many men were attacked, and there were several deaths.'⁴ With a daily average of 635, there were 34 deaths, of which 23 were attributed to beri-beri. The case-incidence was 332, the mortality from it 29 per 1,000.

Though severe, this rate was less bad than that of 1880. Part of the reduction may have been due to the dhal, but a further 'change was introduced in the diet of the prisoners,' Simon says,⁵ 'during the last quarter of the year, consisting in the allowance of a ration of black beans instead of a portion of the rice.'

Rowell⁶ says this addition of black beans was of 'good effect.'

¹ Straits Settlements Blue-Book, p. 40, para. 56.

² *Dal* or *dhal*—i.e., pulse husked and split. Various peas, beans, and even vetches and lupines are used as *dhal*, the commonest being the chick-pea, or common gram—*Cicer arietinum*, L.—and the pigeon-pea, *Cajanus indicus*, Spreng. Church ('Food-Grains of India') gives a list of twenty such legumes in extended use. E. van Dieren, who, discussing Rowell's beri-beri records at the Singapore Prison, professes himself driven to guess what is meant by *dholl* ('*Beri-Beri: Eene rijstver giftiging*,' p. 29) may be glad to find his conjecture correct.

³ Straits Settlements Blue-Book, 1886: Annual Medical Report for 1885, in which Rowell reviews the whole history of beri-beri in the prison (para. 108).

⁴ Straits Settlements Blue-Book, 1882: Prisons Report for 1881.

⁵ *Ibid.*, Annual Medical Report, p. 396, para. 36.

⁶ *Ibid.*, 1885, *loc. cit.*

The dietary now was :

	Rice.	Meat.	Fresh Fish.	Salt Fish.	Dhal.	Beans.	Fat.	Vegetable and Adjuncts.
	ozs.	ozs.	ozs.	ozs.	ozs.	ozs.	oz.	ozs.
Long sentences	24	7 ^a	7	5 ^b	2	5	$\frac{1}{2}$	9 $\frac{1}{8}$
Short sentences	22	6 ^a	6	3 ^b	2	5	$\frac{1}{2}$	9 $\frac{1}{8}$

a, Four days a week, beef for Malays, beef and pork alternately for Chinese, mutton twice weekly for Indians.

b, Thrice weekly for Chinese and Malays, four times for Indians.

The daily 'effective' ration (averaged from all the week's allowance) was, for Chinese and Malays :

	Rice.	Meat (including Fresh Fish and Salt).	Fat.	Vegetable and Adjuncts.
	ozs.	ozs.	oz.	ozs.
Long sentences	24	13·14	$\frac{1}{2}$	16·12
Short sentences	22	10·70	$\frac{1}{2}$	16·18

For Indians, the ration of 'meat' contained 9·87 and 7·71 ounces in the two classes.

The differences made at this time (and in earlier years) between the three nationalities in regard to composition of diet are particularly worthy of note, since, slight as they were, they yet profoundly influenced the result—the extent to which the three races respectively were affected by beri-beri. The Chinese, who alone had pork, got most fat in their food, for the pork used in the East is always extremely fatty; both these and the Malay got more proteid stuff (meat of sorts) than did the Tamils. Now, the severity of incidence of the disease on the three races was exactly in the inverse order. The Tamils, who got relatively least meat (and by consequence most rice) suffered most, the Malays next, and the Chinese least. This state of affairs, in striking contrast to what obtains always outside the gaols, continued so long as these differences in the diets were maintained, and ceased when the diets were made alike (in the relative proportion of proteid, starch, and fatty food) for all races, as happened later. There was no other difference in the circumstances of these different races within the gaol at that time which could in any way bring about such a difference in the incidence of beri-beri upon them.¹

The prisoners were now for the first time upon a reasonably-proportioned diet, the effects of which became manifested in the following year.

1882.—At the end of 1881 and the beginning of 1882 new gaol buildings of a model pattern, on an entirely new elevated and well-drained site, were occupied, and it was hoped that with this event the disease would be shaken off. That expectation was not, however, realized, beri-beri recurring as usual, although only after six months'

¹ See text, p. 67, and compare also Bentley's 'Beri-beri,' p. 7.

508 THE CAUSE AND PREVENTION OF BERI-BERI

entire freedom from it—a result which, it might have been supposed, would have at least led to the abandonment by the local observers of all further belief in local 'miasmata' as its origin. Simon reports :¹ 'The outbreak of beri-beri occurred early in August, and the last case was admitted on October 23.' In an average strength of 688 prisoners there had been 87 cases, with 23 deaths.

In the epidemics of every previous year the months of November and December had contributed large numbers of cases, and it will be seen this was a feature also in subsequent years. The abrupt cessation of the 1882 epidemic in October, therefore, seems remarkable. But there is the usual explanation :

'In September' of the year, Simon goes on to say in the same report, 'a change was made in the diets of the prison as follows : The ration of salt fish was abolished, and an allowance of beef or pork substituted—the former for Malays and some Indians, the latter for Chinese. For those who do not eat beef, fresh fish has been substituted for the salt fish. A ration of *shamsu* (rice-spirit) was served twice daily to all prisoners who had had beri-beri. While the outbreak was at its height, a pineapple was given each prisoner daily, on the ground of its having proved useful in Riouw Prison. Since the disease stopped, plantain has been substituted for the pine.'

In addition to 23 deaths from beri-beri, there were 30 assigned to other causes in this year.

The dietary now was :

Rice	22 ounces.
Meat	7 ^a "
Fish	7 ^b "
Dhal	2 "
Beans	5 "
Fat	$\frac{1}{2}$ ounce.
Vegetable and adjuncts	16 $\frac{1}{2}$ ounces.

^a, Four times weekly for Malays and Chinese, twice for Indians.

^b, Thrice weekly for all.

For long sentences the rice was 24 ounces.

The daily effective ration was thus for Malays and Chinese :

Rice	22 or 24 ounces.
Meat (including fish)	7 ounces.
Fat	$\frac{1}{2}$ ounce.
Vegetable and adjuncts	16 $\frac{1}{2}$ ounces.

Indians had 2 ounces meat less daily. The proportion of rice in this diet was 55 per cent. of the whole, instead of 71 per cent., as it had been from July, 1880, to September, 1881.

1883.—There were '19 cases only of beri-beri,' Simon says,² 'admitted, of which 5 were primary and 14 secondary cases (relapses). Four cases occurred in prisoners admitted to gaol with the disease. Eight deaths took place—1 in a primary, 7 in secondary cases.'

¹ Straits Settlements Blue-Book, 1883: Annual Medical Report for 1882, p. 222, paras. 95, 96.

² Straits Settlements Blue-Book, 1884: Annual Medical Report for 1883.

1884.—Seven cases were reported—no deaths.

1885.—There were '64 cases of beri-beri, of which 5 were cases remaining over from 1884, and 59 occurring subsequently. There were 4 deaths. . . . In consequence of this outbreak, a change was made in the prison diet, 4½ ounces of wheat-flour being substituted for 8 ounces of rice. The change came into force July 24.'¹

The ration now was :

Rice	14	ounces
Wheat-flour	4½	"
Meat	6	"
Fish	4	"
Vegetables (including dhal, etc.)	14	"

daily for all, the proportion of rice in the whole being 33 per cent.

'Since its adoption,' writes Rowell, 'not only has the general sick-rate fallen, but whilst during the six months preceding its use 55 prisoners had developed beri-beri, of whom 4 died, not a single case of the disease has appeared from the date of its commencement, July 24, to December 31, 1885.'² Rowell adds the interesting fact that the reason for his adoption of wheat-flour was 'based on authentic information, from two or three independent sources, of its efficacy in stopping outbreaks of the disease among native crews on board ship.'

1885 to 1897.—The complete freedom of beri-beri which followed the adoption of this diet continued even longer than the diet was maintained—for *twelve years*.

For whatever reason—possibly the clamour of the convicts for more or bulkier food, possibly for economy—in 1895 the quantity of rice in the diet had again become increased, at the expense of the proteid stuff, and in 1897 beri-beri reappeared.

1897.—Commencing with a few—7—cases in September of 1897, it rose in 1898 to a severe epidemic, which lasted with varying fluctuations down to the spring of 1903.

In 1897 the diets were as follows :³

	Rice.	Meat.	Salt Fish.	Dhal.	Beans.	Wheat- flour.	Fat.	Vegetable and Adjuncts.
Ordinary diet..	OZS. 19 ³	OZS. 6	OZS. 4	OZS. 2	OZS. —	OZS. 4½	OZ. 1	OZS. 10
Penal diet ..	20	—	—	—	5	4½	1	5½

Penal diet only daily for all native prisoners serving under three months, and alternately with *ordinary diet* to all others for the first six months of their sentence, after which *ordinary diet* daily for all.

For those furnishing the great bulk of all the cases of beri-beri which occurred—viz., short sentences of six months, and long sentences of more—the effective daily allowance of the various components works out as follows :

¹ Rowell, *loc. cit.*

² *Ibid.*

³ I have been unable to ascertain the date at which the diets of 1885 became altered.

510 THE CAUSE AND PREVENTION OF BERI-BERI

Rice	19·4 ounces.
Meat (including salt fish)	5·84 ..
Fat	1 ounce.
Vegetables (including dhal, beans, flour, and adjuncts)	14·96 ounces.

The rice thus now formed 46 per cent. of the whole ration.

1898.—Of 4,083 committals, 124 had beri-beri; 18 per cent. of the long sentences suffered. The incidence per 1,000 of daily strength was 124. In this year the allowance of rice was reduced—but at what date does not appear—by 5 ounces.

1899.—This year the 5 ounces previously taken off were restored to the dietary. More cases occurred, and the case-incidence also rose, forming 213 per 1,000 of strength.

1900-1901.—The rice was increased again to 22 ounces, forming 57 per cent. of the whole ration. The case-incidence also rose, being in the former year 266, and in the latter 252 per 1,000.

In the following table the varying proportion of rice in the daily ration, and the fluctuations in the beri-beri rate are compared for the term of years dealt with :

Period.	Rice : Actual Quantity Eaten Daily.	Proportion to Whole Ration.	Beri-beri : Case-incidence per 1,000 of Daily Average Strength of Prisoners.
1869 to 1875 ..	ozs. 28	per cent. 79	(Probably always severe — annual mortality 24 per 1,000)
1875	28	79	921
1876 (Jan. to July)	71 }	159 or more
.. (July to Dec.)	68 }	slight (a few cases)
1877	68	153 or more
1878	71	388
1879 (to Nov.)	71 }	748
.. (Nov., Dec.)	79 }	332
1880 (to July)	79 }	126
.. (July to Dec.) ..	24	71 }	28
1881 (to Sept.)	71 }	10
.. (Sept. to Dec.)	55 }	98
1882	—
1883	—
1884	7 cases (Sept. to Dec.)
1885 (to July)	154
.. (July to Dec.) ..	14	33	213
1886 to 1896	266
1897	19·4	46	252
1898	14·4	41	
1899	19·4	46	
1900	22	57	
1901	

APPENDIX I

511

In the *Selangor Gaols*—Kuala Lumpur ('Old') and Pudo (New)—from 1892 to 1902 the diets were as follows :

DIETS IN SELANGOR GAOLS, 1892-1902 :

	1892.		1893.		1895.		1899.		1900.		1901. f		1902.	
	A.	B.	A.	B.	A.	B.	A.	B.	A.	B.	OZS.	OZS.	A.	B.
Rice ..	21½	21½	14	20	19	20	19	20	21	22	21	20	19	20
Fresh meat ..	5½ ^a	—	6 ^c	—	6	—	6	—	6 ^e	—	6½	—	6	—
Fresh fish ..	—	—	4	—	—	—	—	—	—	—	—	—	—	—
Salt fish ..	—	—	4 ^d	—	4	—	4	—	6 ^f	—	—	—	6 ^f	—
Vegetables (green-stuff) ..	15½	—	7	4	7	4	7	4	7	5	7	—	7	4
Beans ..	6½	—	5	5	—	5	2	5	—	—	—	—	—	—
Towgay i ..	—	—	—	—	—	—	—	—	2	2	2	—	2	2
Dhal ..	—	—	2	—	2	—	—	—	—	—	—	—	—	—
Oil ..	1	—	1	—	1	—	1	—	1	—	1	—	1	—
Curry-stuff ..	1	—	1	—	1	—	1	—	1	—	1	—	1	—
Salt ..	1	1	1	3	1	3	1	—	1	½	—	½	1	½
Pepper ..	—	—	1	—	1	—	1	—	—	—	—	—	—	—
Bread ..	5½	—	—	—	4½	—	—	—	—	—	—	—	—	—
Wheat-flour ..	—	—	4½	4½	—	4½	4½	—	—	—	—	—	—	—

A.—Ordinary diet, given to all long-sentence prisoners daily, after first six months of incarceration ; and to all other criminal prisoners (except those serving under three months) on four days a week, alternating with

B.—Penal diet, given to all short-sentence prisoners whose sentences are under three months, daily ; and to others thrice a week.

a, Thrice weekly ; b, four days a week ; c, once a week ; d, six days weekly ; e, thrice weekly ; f, four times weekly ; g, Dr. H. Wright's experimental period, from May 3, 1901, to April 1, 1902 (see p. 172), all inmates on one diet ; h, butchers' meat for all except Sikhs, who got two ducks' eggs daily ; i, Towgay. described by Dr. Wright as a 'highly nitrogenized germinating bean' ('Study,' No. 1, para. 291), is a common pea, or gram, such as when used dried and split, is called *dhal*—usually the chick-pea *Cicer arstinum*—allowed to sprout in water before use.

512 THE CAUSE AND PREVENTION OF BERI-BERI

The actual quantity and the proportion of rice in the daily effective ration of those composing the great bulk of the prisoners who got beri-beri in this gaol—namely, those serving between three and six months for the various years appeared as follows :

Year.	Proportion of Rice per Cent. of Whole Diet.	Actual Quantity in Ounces.	Case-incidence of Beri-beri per 1,000 of Strength.
1892	52	21·3	56
1893	41	16·5	{ 14
1894			
1895	46·5	19·4	{ 200
1896			{ 709
1897			{ 767
1898			{ 189
1899	54	19·4	207
1900	59	21·4	315
1901 ¹	54	21	157
1902	59	21·4	483

¹ To May 3, same as in 1900.

APPENDIX II

IN the following tables are shown admissions of Chinese patients for all causes, for beri-beri, and the mortality from beri-beri, at thirty-one district hospitals in the Straits Settlements and Native Malay States between 1881 and 1902.

The tables account for 466,693 admissions of Chinese to various hospitals, among whom were 123,639 cases of beri-beri, with 19,459 deaths.

NOTE.—It is from the figures in these tables that the charts and calculations of the secular fluctuations of beri-beri given in the text have been derived.

Year.	1. Alor Gajah.			2. Bagan Serai, Province Wellesley.			3. Balik Pulau.			4. Batu Gajah.		
	Number of Chinese Patients admitted.	Beri-beri.		Number of Chinese Patients admitted.	Beri-beri.		Number of Chinese Patients admitted.	Beri-beri.		Number of Chinese Patients admitted.	Beri-beri.	
		Cases.	Deaths.		Cases.	Deaths.		Cases.	Deaths.		Cases.	Deaths.
1881	—	—	—	—	—	—	—	—	—	—	—	—
1882	—	—	—	—	—	—	—	—	—	—	—	—
1883	—	—	—	—	—	—	96	11	3	—	—	—
1884	—	—	—	—	—	—	344	53	8	—	—	—
1885	—	—	—	—	—	—	407	102	22	—	—	—
1886	—	—	—	—	—	—	322	58	5	—	—	—
1887	—	—	—	—	—	—	436	41	4	—	—	—
1888	—	—	—	—	—	—	311	18	2	—	—	—
1889	—	—	—	—	—	—	435	45	6	—	—	—
1890	208	36	6	—	—	—	383	15	6	—	—	—
1891	430	59	6	—	—	—	368	16	2	—	—	—
1892	498	41	4	—	—	—	382	1	1	—	—	—
1893	731	98	4	—	—	—	365	18	7	2,487	1,039	112
1894	734	133	5	—	—	—	304	11	3	2,230	693	114
1895	1,171	180	7	—	—	—	429	52	22	2,913	1,154	93
1896	1,127	195	18	867	81	4	480	69	25	3,474	1,497	151
1897	1,112	255	18	1,490	62	1	472	32	13	2,976	1,016	64
1898	678	60	—	1,514	14	3	416	6	1	2,766	993	72
1899	468	39	5	1,282	25	3	356	28	3	2,282	702	86
1900	429	47	5	1,613	8	2	361	33	10	4,021	2,249	362
1901	569	25	6	2,236	12	5	357	14	5	3,181	1,411	240
1902	310	23	2	1,519	12	2	343	1	—	2,622	1,070	245
Totals	8,365	1,189	86	10,521	214	20	7,369	624	148	28,952	11,124	1,539

APPENDIX II

515

Year.	5. Butterworth.			6. Bukit Mertajam.			7. Durian Daun, Malacca.			8. Gopeng.		
	Number of Chinese Patients admitted.	Beri-beri.		Number of Chinese Patients admitted.	Beri-beri.		Number of Chinese Patients admitted.	Beri-beri.		Number of Chinese Patients admitted.	Beri-beri.	
		Cases.	Deaths.		Cases.	Deaths.		Cases.	Deaths.		Cases.	Deaths.
1881	—	—	—	—	—	—	841	55	6	—	—	—
1882	—	—	—	—	—	—	1,342	217	28	—	—	—
1883	—	—	—	—	—	—	950	95	9	—	—	—
1884	—	—	—	—	—	—	918	103	40	—	—	—
1885	—	—	—	—	—	—	1,020	272	77	—	—	—
1886	—	—	—	—	—	—	950	193	46	—	—	—
1887	—	—	—	—	—	—	1,100	380	59	—	—	—
1888	—	—	—	—	—	—	2,000	842	74	—	—	—
1889	—	—	—	—	—	—	2,207	1,007	225	—	—	—
1890	—	—	—	—	—	—	1,184	461	132	—	—	—
1891	—	—	—	—	—	—	1,695	462	55	—	—	—
1892	130	11	2	—	—	—	1,779	604	33	—	—	—
1893	122	—	—	—	—	—	2,300	650	44	—	—	—
1894	109	2	—	—	—	—	1,942	405	23	—	—	—
1895	272	35	7	422	2	—	1,968	370	59	2,754	971	142
1896	459	81	13	667	35	5	2,171	508	83	2,595	959	130
1897	242	18	5	484	40	13	2,015	572	92	2,086	751	102
1898	263	6	1	386	5	—	2,070	180	15	1,714	494	51
1899	152	4	2	217	4	3	2,245	362	65	1,503	380	35
1900	147	8	2	237	7	2	2,280	246	64	2,015	670	112
1901	184	13	7	317	15	4	1,868	167	45	2,195	584	54
1902	117	9	3	291	18	2	1,845	147	30	2,081	481	38
Totals	2,197	187	42	3,702	140	30	36,780	8,247	1,304	16,943	5,290	664

516 THE CAUSE AND PREVENTION OF BERI-BERI

Year.	9. Ipoh.				10. Jasin.				11. Jelebu.				12. Kajang.			
	Number of Chinese Patients admitted.	Beri-beri.		Number of Chinese Patients admitted.	Cases.	Deaths.	Beri-beri.		Number of Chinese Patients admitted.	Cases.	Deaths.	Number of Chinese Patients admitted.	Beri-beri.		Number of Chinese Patients admitted.	Cases.
		Cases.	Deaths.													
1881	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
1882	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
1883	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
1884	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
1885	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
1886	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
1887	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
1888	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
1889	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
1890	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
1891	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
1892	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
1893	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
1894	2,851	1,316	302	697	54	3	21	2	183	47	6	47	17	4	47	4
1895	3,168	1,535	268	851	111	7	32	7	268	92	13	92	49	11	147	11
1896	3,186	1,332	217	851	111	7	41	4	513	133	27	133	47	15	91	15
1897	3,442	1,245	199	1,276	190	14	64	4	725	166	27	166	87	16	192	16
1898	3,106	953	118	1,154	229	8	54	3	301	238	22	238	180	14	349	14
1899	2,870	828	95	1,076	152	20	111	7	430	117	8	117	222	33	353	33
1900	3,841	1,637	256	1,041	163	47	190	14	374	173	9	173	219	23	343	23
1901	2,983	939	153	838	88	31	229	8	342	151	2	151	212	35	328	35
1902	2,564	709	90	493	37	18	152	20	206	47	—	47	345	67	1,021	67
Totals	28,011	10,494	1,698	9,310	1,926	184	1,926	184	4,268	1,755	144	1,755	2,607	261	6,475	261

APPENDIX II

517

Year.	13. Kampar.			14. Klang.			15. Kuala Kangsar.			16. Kuala Kubu.		
	Number of Chinese Patients admitted.	Beriberi.		Number of Chinese Patients admitted.	Beriberi.		Number of Chinese Patients admitted.	Beriberi.		Number of Chinese Patients admitted.	Beriberi.	
		Cases.	Deaths.		Cases.	Deaths.		Cases.	Deaths.		Cases.	Deaths.
1881	—	—	—	—	—	—	—	—	—	—	—	—
1882	—	—	—	—	—	—	—	—	—	—	—	—
1883	—	—	—	43	24	9	—	—	—	—	—	—
1884	—	—	—	91	41	11	—	—	—	—	—	—
1885	—	—	—	126	50	7	—	—	—	—	—	—
1886	—	—	—	201	59	9	380	118	46	—	—	—
1887	—	—	—	252	46	9	489	131	23	—	—	—
1888	—	—	—	237	60	10	341	71	15	193	131	45
1889	—	—	—	261	75	18	318	34	11	430	177	37
1890	—	—	—	266	44	4	216	31	8	635	257	31
1891	—	—	—	122	27	1	207	13	8	460	186	25
1892	—	—	—	164	47	13	355	17	5	677	281	44
1893	—	—	—	202	102	25	449	60	19	620	253	70
1894	—	—	—	179	61	14	587	65	9	641	242	34
1895	—	—	—	217	57	12	595	69	7	1,062	445	37
1896	343	128	9	357	67	13	840	60	6	1,721	780	155
1897	1,134	382	17	405	67	7	1,066	69	2	1,495	524	122
1898	1,267	284	13	416	41	3	681	50	7	1,441	113	27
1899	879	216	15	432	48	7	687	60	8	1,204	133	49
1900	1,543	513	51	465	76	10	773	135	6	1,331	175	56
1901	1,106	340	42	769	48	11	871	163	7	1,357	159	27
1902	1,114	282	26	553	32	5	624	97	9	1,386	185	51
Totals	7,386	2,116	173	5,715	1,048	189	8,879	1,233	196	14,653	3,941	820

518 THE CAUSE AND PREVENTION OF BERI-BERI

Year.	17. Kuala Langat.				18. Kuala Lumpur.				19. Kuala Selangor.				20. Larut.			
	Number of Chinese Patients admitted.		Beri-beri.		Number of Chinese Patients admitted.		Beri-beri.		Number of Chinese Patients admitted.		Beri-beri.		Number of Chinese Patients admitted.		Beri-beri.	
	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.
1881	—	—	—	—	—	—	—	—	—	—	—	—	1,698	—	1,206	518
1882	—	—	—	—	—	—	—	—	—	—	—	—	3,068	—	2,312	649
1883	—	—	—	—	357	58	114	58	—	—	—	—	5,698	—	4,143	684
1884	—	—	—	—	420	53	246	53	—	—	—	—	7,971	—	5,238	318
1885	—	—	—	—	880	69	573	69	—	—	—	—	3,948	—	1,084	63
1886	—	—	—	—	1,916	134	797	134	—	—	—	—	4,285	—	1,151	77
1887	—	—	—	—	4,773	205	1,452	205	—	—	—	—	4,366	—	1,148	83
1888	—	—	—	—	4,832	360	1,786	360	—	—	—	—	4,274	—	758	56
1889	—	—	—	—	3,923	254	1,210	254	—	—	—	—	4,102	—	450	82
1890	—	—	—	—	3,233	217	1,144	217	—	—	—	—	3,210	—	385	23
1891	—	—	—	—	3,424	226	1,326	226	—	—	—	—	3,027	—	127	7
1892	—	—	—	—	3,446	249	1,493	249	—	—	—	—	3,544	—	151	24
1893	—	—	—	—	6,269	611	2,382	611	—	—	—	—	3,557	—	200	44
1894	43	—	1	—	5,293	495	1,716	495	11	—	1	—	3,841	—	193	44
1895	34	—	5	—	5,930	411	1,807	411	27	—	1	—	3,360	—	201	46
1896	23	—	4	—	5,669	394	2,123	394	4	—	3	—	4,042	—	283	54
1897	111	8	30	8	5,341	512	2,218	512	15	—	10	—	3,898	—	240	31
1898	148	6	33	6	5,341	123	804	123	13	—	4	—	3,240	—	213	24
1899	105	6	17	6	2,929	170	805	170	20	—	2	—	2,243	—	172	32
1900	131	10	17	10	2,661	805	805	805	210	—	1	—	2,373	—	380	104
1901	96	8	10	8	3,826	937	937	937	107	—	5	—	2,075	—	281	68
1902	95	3	10	3	3,764	696	696	696	34	—	1	—	2,026	—	180	43
1902	83	10	14	10	3,498	839	839	839	39	—	—	—	—	—	—	—
Totals	869	51	141	51	72,384	4,945	24,469	4,945	480	28	9	28	80,856	3,074	20,456	3,074

¹ Includes all nationalities.

APPENDIX II

519

Year.	21. Perit Buntar.			22. Penang (Pauper).			23. Rawang.			24. Selama.		
	Number of Chinese Patients admitted.	Beriberi.		Number of Chinese Patients admitted.	Beriberi.		Number of Chinese Patients admitted.	Beriberi.		Number of Chinese Patients admitted.	Beriberi.	
		Cases.	Deaths.		Cases.	Deaths.		Cases.	Deaths.		Cases.	Deaths.
1881	—	—	—	—	—	—	—	—	—	—	—	—
1882	—	—	—	—	—	—	—	—	—	—	—	—
1883	—	—	—	—	—	—	—	—	—	—	—	—
1884	—	—	—	1,186	238	54	—	—	—	—	—	—
1885	—	—	—	1,341	420	172	—	—	—	—	—	—
1886	—	—	—	1,388	477	100	—	—	—	—	—	—
1887	—	—	—	1,084	275	54	—	—	—	—	—	—
1888	—	—	—	1,358	190	56	—	—	—	—	—	—
1889	477	65	6	2,422	280	44	19	2	1	—	—	—
1890	390	70	11	2,470	404	77	185	81	21	—	—	—
1891	657	48	6	2,621	239	61	434	157	47	—	—	—
1892	974	10	1	2,059	126	33	841	303	61	58	7	6
1893	1,091	43	5	2,110	189	52	2,064	868	168	48	6	1
1894	1,919	76	7	3,143	283	46	1,485	512	95	32	4	—
1895	1,769	131	14	3,557	316	79	1,254	430	110	107	16	3
1896	2,181	93	11	3,872	274	103	1,361	547	148	31	8	3
1897	1,018	12	1	3,089	335	116	1,327	502	91	20	1	—
1898	1,043	8	1	2,829	216	71	569	159	8	21	1	—
1899	651	45	3	2,203	229	78	388	114	13	25	—	—
1900	536	3	—	2,513	359	98	750	191	4	19	4	1
1901	651	8	2	2,655	380	134	1,107	228	18	28	5	—
1902	523	2	1	2,461	314	121	908	164	13	29	7	2
Totals	1,583	612	69	44,361	5,521	1,549	12,692	4,250	805	418	59	16

¹ Includes all nationalities.

520 THE CAUSE AND PREVENTION OF BERI-BERI

Year.	25. Seremban.				26. Serendah.				27. Sungai Bakap.				28. Sungai Besi.			
	Number of Chinese Patients admitted.	Beri-beri.		Deaths.	Number of Chinese Patients admitted.	Beri-beri.		Deaths.	Number of Chinese Patients admitted.	Beri-beri.		Deaths.	Number of Chinese Patients admitted.	Beri-beri.		Deaths.
		Cases.	Deaths.			Cases.	Deaths.			Cases.	Deaths.			Cases.	Deaths.	
1881	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
1882	90	56	27	—	—	—	—	—	409	149	14	—	—	—	—	—
1883	41	19	6	—	—	—	—	—	172	33	4	—	—	—	—	—
1884	116	48	11	—	—	—	—	—	123	19	5	—	—	—	—	—
1885	116	62	16	—	—	—	—	—	203	90	2	—	—	—	—	—
1886	161	99	16	—	—	—	—	—	276	133	8	—	—	—	—	—
1887	137	118	33	—	—	—	—	—	150	33	—	—	—	—	—	—
1888	368	219	39	—	—	—	—	—	181	52	3	—	—	—	—	—
1889	785	405	59	—	—	—	—	—	160	59	1	—	—	—	—	—
1890	813	359	40	—	—	—	—	—	109	21	2	—	—	—	—	—
1891	596	268	31	—	—	—	—	—	88	10	2	—	—	—	—	—
1892	501	169	27	—	—	—	—	—	119	10	1	—	—	—	—	—
1893	645	308	17	—	—	—	—	—	161	7	—	—	—	—	—	—
1894	590	277	30	—	162	25	3	—	207	7	1	—	—	—	—	—
1895	697	378	53	—	1,283	431	48	—	214	9	3	—	260	187	26	—
1896	956	588	78	—	2,096	985	152	—	277	9	4	—	395	227	31	—
1897	774	326	55	—	1,552	895	170	—	162	2	—	—	255	159	31	—
1898	731	190	9	—	1,294	348	45	—	216	3	—	—	221	75	9	—
1899	970	463	8	—	1,046	197	21	—	110	2	—	—	253	66	11	—
1900	1,413	772	8	—	1,102	206	25	—	127	—	—	—	159	70	11	—
1901	1,333	688	37	—	1,230	183	17	—	87	—	—	—	167	75	8	—
1902	992	340	35	—	1,055	193	12	—	83	24	—	—	176	75	21	—
Totals	12,825	6,080	605	—	10,820	3,261	493	—	3,634	650	50	—	1,886	934	148	—

APPENDIX II

521

Year.	29. Tampah.				30. Tapah.				31. Teluk Anson.			
	Number of Chinese Patients admitted.	Beri-beri.		Deaths.	Number of Chinese Patients admitted.	Beri-beri.		Deaths.	Number of Chinese Patients admitted.	Beri-beri.		Deaths.
		Cases.	Deaths.			Cases.	Deaths.			Cases.	Deaths.	
1881	—	—	—	—	—	—	—	—	—	—	—	—
1882	—	—	—	—	—	—	—	—	—	—	—	—
1883	—	—	—	—	—	—	—	—	—	—	—	—
1884	—	—	—	—	—	—	—	—	—	—	—	—
1885	—	—	—	—	—	—	—	—	—	—	—	—
1886	—	—	—	—	—	—	—	—	—	—	—	—
1887	—	—	—	—	—	—	—	—	360	189	19	6
1888	—	—	—	—	—	—	—	—	362	81	12	6
1889	—	—	—	—	—	—	—	—	367	130	3	6
1890	—	—	—	—	—	—	—	—	283	86	3	6
1891	217	32	8	—	633	72	6	—	189	37	10	10
1892	382	90	5	—	1,427	129	4	—	226	28	7	7
1893	559	118	8	—	1,680	181	29	—	406	41	7	7
1894	676	150	3	—	1,302	134	38	—	632	52	13	13
1895	784	167	9	—	1,655	196	27	—	515	224	8	8
1896	427	129	11	—	1,633	177	23	—	378	43	5	5
1897	469	24	18	—	1,377	283	28	—	467	54	8	8
1898	537	21	3	—	1,089	355	23	—	305	65	2	2
1899	380	52	4	—	973	247	42	—	306	36	6	6
1900	325	36	6	—	1,142	416	77	—	260	35	11	11
1901	271	21	8	—	1,252	362	125	—	228	51	26	26
1902	289	33	8	—	1,165	324	71	—	212	41	14	14
Totals	5,316	873	91	—	13,328	2,685	493	—	5,695	1,226	163	163

¹ Includes all nationalities.

LITERATURE¹

- ADRIANI, P. : 'En de rijstvergift-hypothese,' *Geneesk. Courant*, vol. lvii. (1903), p. 181.
- ALLBUTT, T. C. : Art. 'Grain-poisoning,' in 'A System of Medicine,' vol. ii. (1900), p. 792.
- ALVARENGA, COSTA : 'Symptomatologie et pathogénie du béri-béri,' Lisbon, 1875.
- ANDERSON, A. R. S. : 'Beri-beri on the R.I.M. Surveying Ships *Investigator* and *Nancowry*,' *Ind. Med. Gaz.*, vol. xxxvi. (1901), p. 330.
- ANDERSON, W. : (1) 'On Kakké, or the Beri-beri of Japan,' *St. Thomas' Hospital Reports*, N.S., vol. vii. (1876), p. 5 ; vol. viii. (1877), p. 247. (2) 'Lectures on Kakké,' Yokohama, 1879.
- ANDRIEUX : 'Une Epidémie de Beri-beri à Poulo-Condore,' *Ann. d'hygiène et de Méd. Col.*, 1902, No. 2.
- ASHMEAD, A. S. : (1) 'Investigation of the Outbreak of Beri-beri on board the barque *Paz* from Ceylon,' *Medical News*, vol. lxiii. (1893), p. 169. (2) 'A New Proof that Beri-beri is due to Carbonic Poisoning,' *Medical Record*, vol. xlv. (1894), p. 461. (3) 'Beri-beri on the barque *Robert S. Patterson* at Perth Amboy, N.J.,' *Medical Record*, vol. xlv. (1894), p. 652. (4) *Sei-i-kwai*, August 31, 1903.
- AZEVEDO : 'Beri-beri in Brazil,' *Deutsch. Mediz. Zeit.*, June 21, 1890, and August 12, 1891.
- BAELZ, B. : (1) 'Ueber das Verhältniss der multiplen peripherischen Neuritis zur Beriberi,' *Zeitschr. f. klin. Med.*, vol. iv. (1882), p. 616. (2) 'Ueber die in Japan vorkommenden Infektionskrankheiten,' *Mitt. der deutsch. Ges. f. Natur- und Völkerkunde Ostasiens*, Tokio, 1882, p. 295 ; Transl. in *Arch. de Méd. Nav.*, vol. xli. (1884), p. 330. (3) 'Behandlung der Beri-beri,' *Penzoldt und Stintring Hand. der spec. Therap. inn. Krankheiten*, vol. i. (1896), p. 668.
- BAILEY, J. W. : 'Beri-beri: a Clinical Study' (Japanese in Vancouver), *North-West Medicine*, February, 1903 ; abstract in *Journal of American Medical Association*, 1903, i., p. 1324.
- BAKER, O. : Edit. on 'Beri-beri in Rangoon,' *British Medical Journal*, 1895. ii., p. 792.

¹ I have included a few references to works on peripheral neuritis from arsenic and other causes, on grain-intoxications, and on parasites of corn. Corre, Fiebig, and Scheube have good bibliographies.

- BARRY, C. C. : (1) 'Notes on Beri-beri in Rangoon,' *Indian Medical Gazette*, vol. xxxv. (1900), p. 343. (2) 'Beri-beri among Tamils in Rangoon,' *Indian Medical Gazette*, vol. xxxvi. (1901), p. 196.
- BENTLEY, A. J. M. : 'Beri-beri : its Etiology, Symptoms, Treatment, and Pathology,' Edinburgh, 1893.
- BERTRAND, G. : 'Sur la recherche et sur l'existence de l'arsenic dans la série animale,' *Ann. de l'Inst. Past.*, vol. xvi. (1902), p. 553 ; vol. xvii. (1903), p. 1 ; *Comptes Rendus*, vol. cxxxiv. (1902), p. 1434.
- BIDIE, G. : 'On the Geographical Distribution of Disease in Southern India,' *British Medical Journal*, 1889, ii., p. 113.
- BIRGE, W. S. : 'Cases of Beri-beri occurring in the Provincetown Grand Bank Fishing Fleet,' *Boston Medical Surgical Journal*, vol. cxxiii. (1890), p. 464.
- BLAISE, H. : 'L'étiologie de la lathyrisme médullaire spasmodique en Algérie,' Algiers, 1900.
- BOARD OF TRADE JOURNAL, THE : Vol. xl. (1903), p. 276, 'Rice Imports into Brazil.'
- BOECK VAN : Art. 'Ergotism,' in 'Ziemmsen's Cyclop. of Med.,' vol. xvii. (1878), p. 891.
- BOLTON, W. G. : 'An Epidemic of Beri-beri at Diego-Garcia,' *Journal of Tropical Medicine*, vol. v. (1902), p. 248.
- BONDURANT, E. M. : (1) 'Report of Thirteen Cases of Multiple Neuritis occurring among Insane Patients,' *Medical News*, vol. lxix. (1896), p. 365. (2) 'Endemic Multiple Neuritis (Beri-beri),' *New York Medical Journal*, vol. lxvi. (1897), pp. 685, 728.
- BÖNNING, H. C. : 'Eight Cases of Beri-beri,' *American Journal of Medical Science*, vol. cvii. (1894), p. 544.
- BOUDIN : 'Géog. et stat. méd. et des malad. endémiques,' 1857.
- BRADDON, W. L. : (1) 'Dochmia and Beri-beri,' Singapore, 1893. (2) 'The Etiology of Beri-beri ; the Effect of Certain Drugs in Beri-beri,' *Federated Malay States Medical Archives*, No. 1 (1900). (3) 'On the Probable Causation of Beri-beri by a Toxin conveyed in Rice,' *Selangor Government Press*, 1901. (4) 'The Light of Local Experience on the Rice Theory of Beri-beri : Where it Fails,' *Taiping Government Press*, 1902.
- BRAULT, J. : 'Traité pratique des maladies des pays chauds et tropicaux' (1900), Art. 'Beri-beri,' p. 580.
- BREFELD : 'Der Reisbrand und der Setaria brand,' *Botan. Central.*, vol. lxxv. (1896), No. 4.
- BRÉMAUD : 'Note sur l'étiologie et l'hygiène préventive du Béri-béri,' *Arch. de Méd. Nav.*, vol. lxxi. (1899), p. 369.
- BRITISH MEDICAL JOURNAL : (1) 'Beri-beri in Mandalay' (1887), ii., p. 1411. (2) 'Cases of Puerperal Neuritis' (1901), i. epit., p. 3 ; (1894) ii. epit., p. 93 ; (1893), i. epit., p. 10.
- BRUNTON, T. L. : 'Disorders of Digestion,' 1886.
- BUCHANAN, W. J. : (1) 'Beri-beri and Rice,' *Lancet*, 1898, ii., p. 577. (2) 'A Note on Lathyrism,' *Journal of Tropical Medicine*, vol. i. (1899), p. 261.
- BULLMORE, C. : 'Beri-beri' (ship cases at Falmouth), *Lancet*, 1900, ii., p. 873.
- BURG, C. L. VAN DER : (1) 'De Geneesheer in Nederlandsch-Indië,' vol. ii., p. 444. (2) 'Statistik der Beri-Beri in het Oost-Indische leger van,

524 THE CAUSE AND PREVENTION OF BERI-BERI

- 1873, tot en met, 1894,' *Ned. Tijdsch. v. Geneesk.* (1896), No. 3.
 (3) 'Le béri-béri et l'alimentation avec du riz,' *Janus*, vol. iii. (1898), pp. 83, 185.
- BUROT AND LEGRANDE: 'Maladies des Marins et épidémies nautiques,' Paris (1886).
- BURY, J.: Art. 'Peripheral Neuritis,' in Allbutt's 'System of Medicine,' vol. vi. (1899), p. 673.
- CAMERON: 'Beri-beri,' *Dublin Journal* (1894), p. 430 (ship cases).
- CARPENTER, P. T.: (1) 'The Clinical Aspects of Beri-beri,' *Journal of Tropical Medicine*, vol. i. (1899), p. 319. (2) 'Observations on the Etiology, Differential Diagnosis, and Treatment of Beri-beri,' *Journal of Tropical Medicine*, vol. ii. (1899), p. 12.
- CARTER, H. J.: 'Beri-beri among the Marines of the Indian Body of H.C. Survey Vessels *Palinurus* and *Norbuddha*,' *Transactions of Bombay Medical and Physiological Society*, No. 8, 1847.
- CARTER, R.: 'Three Cases of Beri-beri,' *British Guiana Medical Journal*, 1902.
- CASEY, J. P. N.: 'An Epidemic of Beri-beri among Boer Prisoners at St. Helena,' *South African Medical Record*, October, 1893.
- CAVALLI, P.: 'Uno caso mortali di Beri-beri' (at Zanzibar), *Ann. di Med. Nav.*, 1897.
- CHAMBERS: 'Skin Lesions in Beri-beri,' *Geneesk. Tijdschr. v. Ned.-Ind.* vol. xx. (1880), p. 45.
- CHAMBERS' ENCYCLOPÆDIA: Art. 'Rice,' vol. viii. (1891), p. 702.
- CHANTEMESSE AND RAMOND: 'Une épidémie de paralysie ascendante chez les aliénées rappelant le béri-béri,' *Ann. de l'Inst. Past.*, vol. xii. (1898), p. 574.
- CHEVERS, N.: 'Beri-beri in India,' *Lancet*, April 5, 1884.
- CHRISTOPHERSON, C. J.: 'Case of Peripheral Neuritis, probably rice produced, simulating Beri-beri, from Omdurman, Soudan,' *Journal of Tropical Medicine*, vol. vi. (1903), p. 154.
- CHURCH, A. H.: 'Food-grains of India,' London, 1886.
- CLARK, F.: 'Beri-beri' (in children in Hong-Kong), *British Medical Journal* (1900), i., p. 1152.
- CLEMOW, F. G.: 'The Geography of Disease,' Cambridge, 1903.
- COMBE, J. DE LA: 'Morbidity et mortalité d'un convoi d'immigrants japonais en Nouvelle-Calédonie en 1901-1903,' *Ann. d'hyg. et de Méd. Col.*, 1904, t. vii., p. 326.
- COMMISSION ON ARSENICAL POISONING, REPORT OF THE ROYAL. 1903.
- CONGRÈS NATIONALE D'HYGIÈNE ET DE CLIMATOLOGIE MÉDICALE DE LA BELGIQUE ET DU CONGO, *Comptes Rendus de*, vol. ii. (1897), p. 472.
- CORLETTE, C.: (1) 'Beri-beri in Australia,' *British Medical Journal* (1895), ii., p. 800. (2) 'The Etiology of Beri-beri,' *British Medical Journal* (1897), ii., p. 680.
- CORNEY, HIRSCH, AND JOYNT: 'Beri-beri in Japanese Immigrants,' *Report of Legislative Council of Fiji*, 1896.
- CORRE: 'Traite clinique et pratique des maladies des pays chauds,' Paris, 1887, pp. 152-249.
- CREIGHTON, C.: Art. 'Pellagra,' in 'Encyclopædia Britannica,' ed. x., vol. xviii., p. 185.

- CROMBIE, A. : 'Acute Œdema in Deccan,' *Indian Medical Gazette*, vol. xiv. (1879), p. 90.
- CROSTHWAITE, W. S. : 'The Wet-pack in Beri-beri,' *British Medical Journal* (1898), i., p. 1258.
- DANGERFIELD, H. V. : 'Le Beri-beri,' Paris, 1905.
- DAÜBLER, K. : (1) 'Die Beriberi Krankheit,' *Wien. Klin. Rundsch.* (1896), pp. 677, 702, 721. (2) 'Die Beriberi Krankheit,' *Virchow's Archiv*, clii. (1898), p. 218. (3) 'Die grundzuge der tropen-hygiene,' Munich, 1895.
- DAVIDSON, A. : 'Epidemic Dropsy,' *Edinburgh Medical Journal*, August, 1881.
- DEBLÈNE : 'Essai de géographie médicale de Nessi-Be,' 'Thèse de Paris,' 1883.
- DÉCHAMBRE, A. : Art. 'Beri-beri,' in 'Dictionnaire Encyclopædique des Sciences Médicales.'
- DÉCHAMBRE AND CUROT on 'Equine infectious mycotic paraplegia': ref. *Veterinary Journal*, March, 1904.
- DELÉPINE, S. : (1) 'Report on Samples of Beer, Brewing, and other Materials,' Salford, 1901. (2) With TATTERSALL, C. H. : 'Demonstration of Arsenic in Beer and its Source,' *British Medical Journal* (1900), ii., p. 1587.
- DIEREN, E. VAN : (1) 'Nogmaals de beri-beri Kwestie,' Arnheim, 1888. (2) 'Beri-beri eene rijstvergiftiging,' Amsterdam, 1897. (3) 'Kantteekeningen op Dr. Vorderman's beri-beri Rapport en nog jets,' 1897. (4) *Geneesk. Tijdschr. v. Ned.-Ind.*, xxxvii. (1897), p. 545.
- DOMENICO, M. : 'Sulle alterazioni del sistema nervosa nell' intossicazione latrica,' *Giorn. de Med. Leg.*, 1898.
- DURHAM, H. E. : 'Notes on Beri-beri in the Malay Peninsula and on Christmas Island (Indian Ocean),' *Journal of Hygiene*, vol. iv. (1904), p. 112.
- DYKES, C. : 'Note on an Outbreak of True Beri-beri in an Assam Gaol,' *Indian Medical Gazette*, June, 1904.
- EBBELL, F. : 'Beri-beri's Aetiologie,' *Norsk Mag. fer Laeger*. August, 1901, p. 958.
- EECKE VAN : *Geneesk. Tijdschr. v. Ned.-Ind.*, vol. xxvii. (1887), p. 71.
- EHLERS, E. : 'L'Ergotisme,' Paris, (n.d.).
- EIJKMAN, C. : (1) 'Polyneuritis bij hoeuveren,' *Geneesk. Tijdschr. v. Ned.-Ind.*, vol. xxx., 1890. (2) 'Verhandeling over de Polyneuritis der Kippen,' *Ned. Tijdschr. v. Geneesk.*, October 20, 1896. (3) 'Polyneuritis bij Hoenders; nieuwe bijdrage tot de aetiologie der Ziekte,' *Jaarverslag van Lab. v. Path. Anat. en Bakt. te Weltevreden over het jaar*, 1895; Batavia, 1896. (4) *Soerabayasch Handelsblad*, March 24, 1897. (5) 'Note sur la prophylaxis du béri-béri,' *Janus*, vol. ii. (1897), p. 23. (6) 'Nogmaals beri-beri en voeding,' *Geneesk. Tijdschr. v. Ned.-Ind.*, vol. xxxviii. (1898), p. 277.
- ELDRIDGE, S. : 'Beri-beri, or the Kakké of Japan,' *Pacific Medical and Surgical Journal*, December, 1880, and January, 1881.
- ELLIS, W. G. : (1) 'A Contribution to the Pathology of Beri-beri,' *Lancet*, 1898, ii., p. 985. (2) 'Annual Medical Report for 1898,' in *Straits Settlements Government Gazette*. (3) 'The Micrococcus of Beri-beri,'

526 THE CAUSE AND PREVENTION OF BERI-BERI

- Lancet*, 1899, i., p. 1662. (4) 'The Etiology of Beri-beri,' *British Medical Journal* (1903), ii., p. 1,268.
- ENCYCLOPÆDIA BRITANNICA: Arts. (1) 'Beri-beri,' (2) 'Brazil,' (3) 'Brewing,' (4) 'Pellagra,' (5) 'Rice,' (6) 'Shipping.'
- ERICHSEN, STIAN: 'Undersegelse em Beri-beri,' *Tidsh. f. den Norsk Laegefelk*, No. 16, 1899.
- ESCOVAR: 'Note on a Case of Beri-beri' (in Panama), *Lancet*, July 19, 1903.
- EYRE: 'Beri-beri in the 28th Regiment Madras Infantry,' *Indian Medical Gazette*, January, 1900, p. 17.
- FAYRER, J.: 'On an Epidemic of Beri-beri,' *Medical Times and Gazette*, 1880, p. 631.
- FÉRIS: 'Le Beri-beri d'après les travaux brésiliens,' *Arch. de Méd. Nav.*, vol. xxxvii. (1882), p. 466; vol. xxxviii. (1882), p. 50.
- FIEBIG, M.: 'Beri-beri ender de dessabovelking in Nederlandsch-Indië,' *Geneesk. Tijdschr. v. Ned.-Ind.*, vol. xxix. (1889), Nos. 2, 3; vol. xxx., p. 432.
- FISCHER, G.: 'Mijne laatste woord aan den Heer E. van Dieren,' *Soerabaya*, 1898.
- FOWLER, J. F. S.: 'Does Beri-beri exist in this Colony?' *British Guiana Medical Annual*, No. xi. (1899), p. 8.
- FREEMAN, F. M.: 'The Seed Fungus of the Darnel,' *Philosophical Transactions*, B. cxcvi. (1903), p. 1.
- GAUTIER, A.: 'Sur l'existence normale de l'arsenic chez les animaux, et sa localisation dans certains organes,' *Comptes rendus de l'Acad. des Sc.*, vol. cxxix. (1899), p. 929; vol. cxxx. (1900), p. 284; vol. cxxxi. (1900), p. 361.
- GAYET: 'Du Beri-beri' (at Puolo Condore), *Arch. de Méd. Nav.*, vol. xlii. (1884), pp. 161, 241.
- GELPKE: 'Etiology of Beri-beri' (1) *Geneesk. Tijdschr. v. N.-I.*, d. xix., p. 273; (2) *ibid.*, d. xxiii., p. 41; (3) *ibid.*, d. xxvii., p. 22; (4) 'Beitrag zur Bestreitung der Beri-beri,' *ibid.*, d. xxx., p. 150 (1890). (5) 'Über die aetiologie der Beri-beri,' *Geneesk. Tijdschr. v. Ned.-Ind.*, vol. xxxvii. (1897), p. 108.
- GERRARD, P. N.: (1) 'The Influence of Rainfall on Beri-beri,' *Lancet* (1899), i., p. 367. (2) 'Beri-beri and its Symptomatic Treatment,' London, 1904.
- GIBSON, R. MACL.: 'Beri-beri in Hong-Kong, with Special Reference to the Records of the Alice Memorial and Nethersole Hospitals, and with Notes of two Years' Experience of the Disease,' *Journal Tropical Medicine*, vol. iv. (1901), pp. 96, 111.
- GIBSON, G. A.: Arts. 'Peripheral Neuritis,' and 'Ergotism,' in 'A Text-book of Medicine,' Edinburgh, 1901.
- GLOGNER, M.: (1) 'Eine weiterer Beitrag zur Ätiologie der multiplen Neuritis in den Tropen,' *Virchow's Archiv*, cxli. (1895), p. 401. (2) 'Über die klinischen formen der Beri-beri Krankheit,' *Virchow's Archiv*, cxlvi. (1896), p. 129. (3) 'Neuere untersuchungen über die Beri-beri Krankheit,' *Archiv für Schiffs und Tropen Hygiene*, i. (1897), pp. 46, 125.

- GORKOM, W. J. VAN : (1) 'De beri-beri kwestie. Vergiftiging of infectie,' *De Indische Gids*, November, 1897. (2) 'Beri-beri in de gevangenissen op Java,' *Geneesk. Tijdschr. v. Ned.-Ind.*, vol. xxxviii. (1898), p. 709 ; vol. xxxix. (1899), pp. 366, 460.
- GOWERS, W. R. : (1) 'Diseases of the Nervous System,' second edition, 1893. (2) 'Accidental Sources of Arsenic-Poisoning,' *Proceedings of the Medical Society*, 1880.
- GRAHAM : 'Beri-beri at Sydney,' *Australasian Medical Gazette*, November 15, 1893.
- GRALL, PORÉE, AND VINCENT : 'Beri-beri en Nouvelle-Calédonie,' *Arch. de Méd. Nav.*, vol. lxiii. (1895), pp. 134, 187, 260.
- GRIJNS : (1) *Deut. Med. Woch.*, 1890, p. 949. (2) 'Over polyneuritis gallinarum . . .', (3) *Mededeelingen v. h. Lab. Path. Anat. en Bakt. te Weltevreden*, 1900. (4) 'Beri-beri on rijstveeding,' *Gen. Tijd. v. N.-I.*, d. xli. (1901).
- GRIMM, F. : (1) 'Ueber Kakke auf Hokkaido,' *Deutsch. Med. Woch.*, 1890, p. 949. (2) 'Klinische Beobachtungen über Beri-beri,' Berlin, 1897. (3) 'Ueber beri-beri,' *Deutsch. Med. Woch.*, 1898, p. 460.
- GUERIN, M. P. : 'Sur la presence d'un champignon dans l'ivraie,' *Journ. de Bot.*, vol. xii. (1898), p. 230.
- GUIOL : 'Beri-beri at Nessi-Be,' *Arch. de Méd. Nav. et Col.*, October, 1882, p. 273.
- HAGEN : 'Du béri-béri à la Nouvelle-Calédonie, et de quelques observations tendant à prouver son caractère contagieuse,' *Rev. Méd. de l'Est.*, 1893, p. 42.
- HANAUSEK, T. F. : 'Vorläufige mitt. über den von A. Vogl in der Frucht van Lolium temulentum entdecktem Pilz,' *Ber. der Deutsch. Bot. Ges.*, vol. xvi. (1898), p. 203.
- HARTIGAN, W. : 'The Etiology of Beri-beri' (a place, not a food disease), *British Medical Journal*, 1903, ii., p. 1591.
- HAYNES, T. H. : 'Notes on Beri-beri in the Australian Pearling Fleet, 1883-1887,' *Journal of Tropical Medicine*, vol. ii. (1900), p. 196.
- HAYWARD : 'Beri-beri at Sydney,' *Australasian Medical Gazette*, vol. xxi. (1902), p. 114.
- HEBERSMITH : 'Ship Cases,' *U.S. Marine Hospitals Reports*, 1881.
- HENDLEY, A. G. : 'Lathyrism,' *British Medical Journal*, 1903, ii., p. 707 ; and *Journal of Tropical Medicine*, vol. vi. (1903), p. 359.
- HIROTA, Z. : 'Über die deuch die Milch der an Kakké leidenden frauen verursachte Krankheit der Säuglinge,' *Central. f. inner. Med.*, 1898, p. 385.
- HIRSCH, A. : Art. 'Beri-beri' in 'Hand. d. hist.-geog. Path.,' vol. ii., 1883.
- HODENPIJL, L. P. G. : *Geneesk. Jaarverslag. bet v. den. Gezond. bij de koninkl. Ned. Marine, gedurende 1896-1898*.
- HOSE, C. : (1) *Medical Review*, June, 1901. (2) *British Medical Journal*, October 28, 1905.
- HUILLET : 'Contribution à la géographie médicale de Pondichéri,' *Arch. de Méd. Nav. et Col.*, t. viii., p. 240, December, 1867.
- HUNTER, W. K. : (1) 'A Contribution to the Etiology of Beri-beri,' *Lancet*, 1897, ii., p. 240. (2) 'A Note on the Etiology of Beri-beri,' *Lancet*, 1898, i., p. 1748.

528 THE CAUSE AND PREVENTION OF BERI-BERI

- IRVING: 'Lathyrism in Cattle,' *American Journal of Medical Science*, 1859, p. 113.
- JAMESON, J. S.: 'Beri-beri on an Outward-bound Steamer from Liverpool,' *Medical Press and Circular*, 1893, ii., p. 298.
- JAPP, A.: 'Rice and all About It,' *Good Words*, 1883, pp. 313, 341.
- JEFFERSON, A.: 'A Case of Pernicious Beri-beri,' *British Medical Journal*, 1898, i., p. 1257.
- JONES, L. H.: 'A Case of Recurrent Alcoholic Peripheral Neuritis' (from small doses), *British Medical Journal*, 1901, i., p. 883.
- JÖRGENSEN, A.: 'Les micro-organismes de la Fermentation,' transl. P. Freund, Paris, 1899.
- JOYNT, H. N.: 'The Etiology of Beri-beri' (cases in Japanese coolies imported into Fiji), *Journal of Tropical Medicine*, vol. iv. (1901), p. 141.
- KELYNACK, T. N. and KIRKBY, W.: 'Arsenical Poisoning in Beer Drinkers,' London, 1901.
- KERMORGANT, R.: (1) 'Morbidity et mortalité des trasportées et relégués en Guyane et Nouvelle-Calédonie,' *Ann. d'hyg. et de Méd. Col.*, t. vi., No. 1, March, 1903, p. 185. (2) 'Beri-beri in French Colonies,' *ibid.*, 1903, Appendix, C. iv.
- KESSLER, H. J.: 'Beri-beri geen rijstvergiftiging,' *Geneesk. Tijdschr. v. Ned.-Ind.*, vol. xxxvii. (1897), p. 339.
- KIRCHBERG, E.: 'Trois cas de beri-beri,' *Gas. Med. di Wantes*, December 12, 1893, p. 10; also *Gaz. des Hôp.*, vol. lxvii. (1894), p. 3.
- KITASATO, G.: 'Bemerkungen zu vorstehender Erwiderung (of Pekelharing and Winkler, pp. 77, 277), *Central. f. Bakt.*, vol. iii. (1888), p. 278.
- KLEM, G.: 'More on Beri-beri,' *Norsk Mag. f. Laegefelk*, 1897.
- KLOSSER: 'Specificke ziekteoorzaken bij de koninkl. Ned. Marine,' *Mil. Gen. Arch.*, 1886.
- KNECHT, E., AND DEARDEN, W. F.: 'The Elimination of Arsenic through the Hair and its Relation to Arsenical Poisoning,' *Lancet*, 1901, i., p. 854.
- KOHLBRUGGE, J. H. F.: 'Zu den periodischen Schwankungen der Infektions Krankheiten' (Diphtheria, Beri-beri), *Therapeutisch. Monat.*, 1899, p. 31.
- KOMOTO, T.: 'Scotoma in Beri-berics,' *Sei-i-kwai*, vol. xxii. (1903), p. 109.
- KÖNIGER: 'Ueber epidemisches Auftreten von Beriberi in Manila, 1882-1883,' *Deutsch. Arch. f. klin. Med.*, vol. xxxiv. (1884), p. 419.
- KRONECKER, F.: 'Einiges über die Beriberi in dem Malayischen Archipel,' *Hygienische Rundschau*, No. 18, p. 883, September 15, 1896.
- KRYSINSKI: 'Patholog. und kritische Beiträge zur Mutterkornfrage,' 1890.
- KYNSEY, W.: 'Report on Anæmia, or the Beri-beri of Ceylon,' *British Medical Journal*, October 15, 1887.
- LAACHE, S.: 'On Beri-beri,' *Norsk Mag. f. Laeger.*, 1896.
- LABOULBÈNE, A.: 'Sur un cas de Beri-beri,' *Arch. de Méd. Nav.*, vol. xxx. (1898), p. 372.
- LAOH, T. H.: 'Jets over de etiologic, prophylaxis en therapie der beri-beri,' Batavia, 1903.

- LACERDA, J. B. DE : 'Peste de cadeiras ou epizootia de maranajo suas analogias con o beri-beri,' Rio de Janeiro, 1885, review in *Berl. klin. Woch.*, vol. xxiii. (1886), p. 159 ('Trypanosomiasis').
- LANCET, THE : (1) 'Beri-beri in Newfoundland Fisheries,' 1891, i., p. 326. (2) 'Vital Statistics of Dutch Navy,' 1900, i., p. 255. (3) 'Beri-beri on H.M.S. *Sphinx* at Muscat,' 1900, ii., p. 1165.
- LASNET : 'Rapport sur le béri-béri observée à la Prison Militaire de Dakar durant l'année 1895,' *Arch. de Méd. Nav.*, vol. lxvii. (1897), pp. 138 and 210.
- LAURENT, L. : (1) 'Rôle de l'insuffisance en matières grasses de la ration alimentaire dans l'étiologie du béri-béri,' *Arch. de Méd. Nav.*, vol. lxxi. (1899), p. 194; abstract in *Lancet*, 1899, ii., p. 51. (2) 'Note sur l'épidémie de béri-béri de 1898 à Poulo-Condore,' *Arch. de Méd. Nav.*, vol. lxxii. (1899), p. 140.
- LEATHER : 'Lathyrism,' *Veterinary Journal*, April, 1885.
- LEENT, VAN : (1) 'Mededeelingen over beri-beri,' *Geneesk. Tijdschr. v. Ned.-Ind.*, vol. xx., 1880, Nos. 5 and 6. (2) 'Sur une forme mixte et peu connue de béri-béri et scorbut,' *Arch. de Méd. Nav.*, vol. lxxix. (1903), p. 275.
- LEEUW, DE : 'Mil. Summier Ziekten Rappoort,' *Geneesk. Tijdschr. v. Ned.-Ind.*, vol. xx. (1880), p. 207.
- LEGGE, T. M. : (1) 'Industrial Arsenic-Poisoning,' *British Medical Journal*, 1901, ii., p. 402. (2) 'Report of Royal Commission on Arsenic Poisoning, 1903.'
- LEOPOLD, W. : 'Zur Pathogenese des Beri-beri,' *Berlin. klin. Woch.*, vol. xxix. (1892), p. 66.
- LE ROY DE MERICOURT, A. : (1) Art. 'Béri-béri,' in *Guide du Méd. Pract.* (Valleix), ed. 5, 1866. (2) Art. 'Béri-béri,' in *Dict. Encyclop. des Sc. Méd.* (A. Dechambre). (3) With FONSSAGRIVES : 'Mémoire sur la caractérisation nosologique de la maladie connue vulgairement dans l'Inde sous le nom de béri-béri,' *Arch. Gen. de Méd.*, Sér. 5, vol. xviii. (1861), p. 257.
- LESLIE, T. T. : 'Notes and Statistics on Administration of Hospitals and Dispensaries in Burmah,' *Calcutta Government Press*, 1899.
- LITTLEFIELD : *Report Surgeon - General Army U.S.A.*, January, February, 1902, 'Beri-beri and Rice in Philippine Prisons,' reference, *Journal American Medical Association*, May 10, 1902, p. 1244.
- LODEWIJKS : 'Hypertrophie en degeneratie van het hart bij Beri-beri,' *Geneesk. Tijdschr. v. Ned.-Ind.*, vol. xix. (1879), p. 17.
- LOKHORST : 'Beri-beri among Native Malays,' *Geneesk. Tijdschr. v. Ned.-Ind.*, vol. xxvii., p. 57.
- LOVELL, F. : 'Report on Acute Anæmic Dropsy in Mauritius,' *Indian Medical Gazette*, vol. xvi. (1881), p. 342; vol. xvii. (1882), p. 25.
- LOWSON, J. A. : 'Beri-beri a Place Disease, not a Food Disease,' *British Medical Journal* (1897) ii., p. 843.
- MACLEAN : 'Beri-beri on Ship *Stirling* at Melbourne,' *Lancet*, 1900, i., p. 138.
- MCLEOD, K. : (1) 'Report on Epidemic Dropsy in Calcutta,' *Indian Medical Gazette*, vol. xvi. (1881), p. 148. (2) 'On Epidemic Dropsy,' *Transactions of the Epidemiological Society, London*, vol. xii., p. 55.
- MACLEOD, N. : 'Can Beri-beri be caused by Food-supplies from Countries

530 THE CAUSE AND PREVENTION OF BERI-BERI

- where Beri-beri is Endemic ?' *British Medical Journal* (1897), ii., pp. 390 and 1459.
- MACLAGAN, C. : 'On the Arsenic-eaters of Styria,' *Edinburgh Medical Journal*, vol. x. (1864), p. 200.
- MACPHAIL : 'Peripheral Neuritis following Influenza,' *American Journal of Insanity*, 1894.
- MAGNAN : 'L'alcoolisme et ses formes diverses,' Paris, 1874.
- MAITLAND, C. B. : 'Peripheral Neuritis following the Soft Sore' (? Syphilis), *British Medical Journal* (1899), i., p. 270.
- MALCOLMSON : 'A Practical Essay on the Symptoms and Treatment of Beri-beri,' Madras, 1835.
- MANSON, P. : (1) Arts. 'Beri-beri' in *Encyclop. Medica* (C. Watson), vol. i. (1899), p. 462 ; 'Manual of Hygiene and Diseases of Warm Climates' (A. Davidson), 1893, p. 452 ; 'System of Medicine' (Allbutt), vol. ii., 1897, p. 439 ; 'Tropical Diseases,' ed. 1903, p. 303. (2) 'The Etiology of Beri-beri,' *Transactions of the Epidemiological Society, London*, vol. xx. (1901), p. 1 ; and *Lancet*, 1901, ii., p. 1391. (3) 'Discussion on Beri-beri,' *British Medical Journal* (1902), ii., p. 530.
- MAZÉ : 'Le Béri-béri,' (epidemy on transport l'Eurydice), *Thèse de Montpellier*, 1852.
- MCCALL : 'Lathyrism,' *The Veterinarian* (1886), p. 789.
- MCCLOSKEY, A. J. : 'Treatment of Beri-beri with Arsenic at the District Hospital, Kuala Lumpur, Selangor,' *Journal of Tropical Medicine* vol. vi. (1903), p. 140.
- McMULLEN, J. C. : 'Five Cases of Beri-beri at Auckland, New Zealand,' *British Medical Journal* (1885), ii., p. 965.
- MEIJER, VAN OVERBECK DE : (1) *Natuur en Geneesk. Arch. v. Ned.-Ind.*, iii. (1846), p. 430. (2) 'Beri-beri,' 's Gravenhage, 1864. (3) *Geneesk. Tijd. v. Zeemacht.*, 1865, p. 1.
- MELARDI, S. : 'Una piccola epidemia di beri-beri sulla R.N. Umbria,' *Ann. di Med. Nav.*, 1898.
- MILROY : 'Notes on the Diseases of Natives of India,' *Transactions London Epidemiological Society*, vol. ii., p. 150.
- MINKS, P. : *Med. Weekblad*, 1894 ('Bacteria in Beri-beri').
- MIURA, M. : (1) 'Beiträg zur . . . path. Anatomie der Kakke,' *Virchow's Archiv*, vol. cxi., 1888, p. 361 ; vol. cxiv., 1888, pp. 341 and 385 ; vol. cxvii. (1891), p. 2 ; vol. cxxiii., 1891, p. 280. (2) 'Beiträge zur Pathologie und Therapie der Kakke,' *Mitt. a. d. Med. Fakult. d. Kaiserl.-jap. Univ. zu Tokio*, vol. iv. (1888), p. 161 ; vol. v. (1889), p. 182.
- MOLITERNI : 'Relazione sanitaria della campagna intorno all' Africa della R.N. Colombo,' *Ann. di Med. Nav.*, November, 1898.
- MOLLOY : 'Is Beri-beri Endemic in Australia ?' *Transactions of the Inter-colonial Medical Congress, Australia*, 1892.
- MOODY, D. W. K. : 'Beri-beri among Lascar Crews on Board Ship,' *British Medical Journal* (1903), i., p. 729.
- MORRIS, H. C. L. : 'The Etiology of Beri-beri in Cocos-Keeling Islands,' *British Medical Journal* (1897), ii., p. 500.
- MOSSÉ, A., and DESTARAC, J. : 'Contribution à l'étude du béri-béri,' *Rev. de Méd.*, vol. xv. (1895), p. 977.
- MOSSE, C. G. D. : *Journal of Hygiene* ('Notes on Beri-beri at St. Helena'), September, 1904.

- MOSSO, J., and MORELLI, J. B. : ' Sur le microbe de béri-béri,' *Gaz. Méd. de Paris* (8), vol. ii. (1893), p. 27.
- MOTT, F. W., and HALLIBURTON, W. D. : (1) ' Note on the Blood in a Case of Beri-beri,' *British Medical Journal* (1899), ii., p. 265. (2) ' The Chemistry of Nerve Degeneration,' *Phil. Trans. Roy. Soc. B.* (1899), p. 1901.
- MURDOCH, J. : ' Lathyrism,' *Science* (New York), vol. viii. (1898), p. 907.
- NAVY, Statistical Report on the Health of the British, 1900 (' Beri-beri on H.M.S. *Forté* ').
- NAUNYN : Art. ' Arsenical Poisoning ' in *Ziemmsen's Cyclop. Med.* (1878), p. 644.
- NEPVEU, G. : (1) ' Nature et Pathologie du Béri-béri,' *Prog. Médic.*, September, 1894. (2) ' Bacilles du Béri-béri,' *Comptes rend. d. l'Acad. des Sc.*, vol. cxxvi. (1898), p. 256 ; and *Marseille Médicale*, August, 1898.
- NESSLER, A. : ' Über einer in der frucht von Lolium Temulentum vorkommenden pilz,' *Ber. Deut. Bot. Ges.*, vol. xvi. (1898), p. 207.
- NIVEN, J. : ' Report of the Medical Officer of Health for Manchester on Outbreak of Arsenic-Poisoning among Beer-Drinkers, 1901.'
- NOCHT : ' Ueber Beri-beri und Skorbut au Bord,' *Hansa*, No. 29, 1900. (2) ' Beri-beri und Skorbut,' *Indistilling* (Report Norwegian Committee), p. 130.
- NORMAN, C. : ' On Beri-beri occurring in Temperate Climates ' (at Richmond Asylum, Dublin), *British Medical Journal* (1898), ii., p. 873 ; (1899), i., p. 409 (skin lesions) ; (1899), ii., p. 686 (diet) ; *Lancet* (1898), ii., p. 377 ; *Dublin Journal of Medical Science*, January 1900, p. 1.
- O'BRIEN, J. : ' Acute Dropsy ' (beri-beri ?), *Indian Medical Gazette*, vol. xiv. (1879), p. 5.
- OERTHMANN : ' Ueber Polyneuritis,' *Allg. Z. f. Psychiatrie*, vol. lvi. (1898), p. 84.
- OGATA : ' Bacteriology of Beri-beri,' *Sei-i-Kwai*, May, 1886.
- OPPENHEIM, H. : ' Diseases of the Nervous System,' trans. F. L. Mayer, second American edition, 1904.
- ORMEROD, E. B. : ' Beri-beri in Queensland,' *Journal of Tropical Medicine* vol. vi. (1903), p. 54.
- OTTINGER : ' Etudes sur les paralysies alcooliques.'
- OUDENHOVEN, H. L. VAN : *Gen. Arch. v. de Zeemacht*, 1874.
- PATON, R. T. : ' Beri-beri at Sydney,' *Australasian Medical Gazette*, November 15, 1894.
- PATON, T. A. : ' Kakké,' *Edinburgh Clinical and Pathological Journal*, 1884.
- PARKER, D. M'N. : ' Case of Death resulting from the Practice of Arsenic Eating,' *Edinburgh Medical Journal*, vol. x. (1864), p. 116.
- PEKELHARING, C. A., and WINKLER, C. : ' Recherches sur la nature et la cause du béri-béri, et sur les moyens de le combattre,' Utrecht, 1888, trans. J. Cantlie, Edinburgh, 1893. See also ' Mitth. über die Beri-Beri,' *Deut. Med. Woch.* (1887), p. 845 ; and *Centr. f. Bakt.*, vol. iii (1888), p. 77.

532 THE CAUSE AND PREVENTION OF BERI-BERI

- PETIT : 'De l'emploi de paddy (riz non décortiqué) dans le béri-béri,' *Ann. d'Hyg. et Méd. Col.*, vol. vi. (1903), p. 98.
- PRAEGER, A. : 'Indische studien,' *Geneesk. Arch. v. de Zeem.*, 1864 and 1871.
- PRILLIEUX, F. : 'Maladies des plantes agricoles,' Paris (n.d.).
- PROUST, A. : 'Du Lathyrism médullaire spasmodique,' *Bull. de l'Acad. Méd.* (1883), Nos. 27 to 29.
- PROUT, W. T. : *British Medical Journal* (1902), ii., p. 838 ('Beri-beri in Negroes').
- PUTNAM, J. J. : (1) 'On Motor Paralysis and other Symptoms of Poisoning from Medicinal Doses of Arsenic,' *Boston Medical and Surgical Journal*, vol. cxviii. (1888), p. 646. (2) 'Multiple Neuritis, or Beri-beri, among Seamen,' *Boston Medical and Surgical Journal*, vol. cxxiii. (1890), pp. 62 and 244.
- RANDELL, P. N. : 'Beri-beri at Ascension,' *British Medical Journal* (June 16, 1900), p. 1505.
- REAUCAR : 'Le béri-béri à Poulo-Condore,' *Thèse de Paris*, 1886.
- RÉBOURGEOIS : 'De la nature infectieuse du béri-béri,' *La Sem. Méd.* (1890), p. 258.
- REES, D. C. : 'Beri-beri a Place Disease, not a Food Disease,' *British Medical Journal* (1897), ii., p. 747.
- REMAK, E., and FLATAU, E. : 'Neuritis und Polyneuritis,' Vienna, 1898.
- RÉMY : 'Notes Médicales sur le Japon,' Paris, 1883.
- REYNOLDS, E. S. : 'Further Observations on Epidemic Arsenical Peripheral Neuritis,' *British Medical Journal* (1900), ii., p. 1769.
- ROBERTS, L. : 'Arsenic in the Hair of Beri-beri Patients,' *British Medical Journal* (1902), i., p. 425.
- ROCHARD, J. : Art. 'Béri-béri' in *Nouv. Dict. de Méd. et de Chirurg. Prat.*
- ROLL : 'Et tiffaelde æf beri-beri,' *Norsk. Magaz. f. Lægevej.*, 1890, 1896.
- ROOSEFELT : (1) *Boston Medical and Surgical Journal*, 1886 ('Epidemy on the *Henry S. Sanford*'); (2) Ship Cases, *Medical Record*, February 19, 1887.
- ROSS, J. : (1) 'Peripheral Neuritis,' *Medical Chronicle*, vol. vii., 1890 ; and (2) 'The Premonitory Symptoms of Alcoholic Paralysis,' *Lancet* (1889), i., p. 1125.
- ROSS, J., and BURY, J. S. : 'On Peripheral Neuritis,' London, 1893.
- ROSS, R. : 'Arsenic in the Hair of Beri-beri Patients from Penang,' *British Medical Journal* (1902), ii., p. 837.
- ROSS, R., and REYNOLDS, E. S. : 'On a Case of Beri-beri (?), possibly due to Arsenic-Poisoning,' *British Medical Journal* (1901), ii., p. 979.
- ROST, E. R. : (1) 'The Cause of Beri-beri,' *Indian Medical Gazette*, vol. xxxv. (1900), p. 458 ; vol. xxxvi. (1901), p. 255 ; vol. xxxvii. (1902), p. 270. (2) *British Medical Journal* (1902), ii., p. 834 (bacillus of beri-beri).
- ROWELL, T. I. : 'Report on Medical Department and Prisons, 1879, 1880, 1881, 1884, and 1886,' *Straits Settlements Government Gazette*.
- RUPERT, J. : 'Ueber beri-beri,' *Deutsch. Archiv f. Klin. Med.*, vol. xxvii. (1880), pp. 95 and 499.
- SAKAKI, J. : (1) 'The Causal Relation between Rice and Beri-beri,' 1892. (2) 'Investigations on Poisonous Rice,' Sei-i-Kwai, March and April, 1903. (3) 'Recherches sur le poison du riz dans l'étiologie du béri-béri,' *La Caducée*, vol. iii. (1903), p. 278.

- SANEYOSHI : 'Rice and Beri-beri,' *Sei-i-Kwai*, April and May, 1901.
- SCHEER, A. VAN DER : 'Een wenschelijke richting van onderzoek naar de oorzaken van beri-beri,' *Geneesk. Tijdschr. v. Ned.-Ind.*, vol. xl. (1900), p. 25; abstract in *Journal of Tropical Medicine*, vol. iii. (1900), p. 96.
- SCHUEBE, B. : (1) 'Die Japanische Kakké,' *Deut. Arch. f. klin. Med.*, vol. xxxi. (1882), p. 141; vol. xxxii. (1883), p. 83. (2) 'Die beri-beri krankheit; eine geographische medizinische studie,' Jena, 1894. (3) 'Die beri-beri epidemien in Richmond Asylum in Dublin,' *Arch. f. Schiffs und Tropen Hyg.*, vi. (1898), p. 329. (4) 'Die Krankheiten der warmen Länder,' ed. 2, Jena, 1900.
- SCHMIDT, P. : 'Zwei fälle von beri-beri an bord eines deutschen Dampfers,' *Munch. Med. Woch.* (1900), p. 191.
- SCHNEIDER : (1) 'Beri-beri,' Soerabaya, 1863. (2) *Gen. Tijdschr.*, xxiii., afl. 4.
- SCHUTTE, W. : 'Beri-beri beschouwd als secondaire pernicieuse anaemie,' Utrecht, 1898.
- SCHUTTELAERE : 'Note sur une épidémie du béri-béri a Diego Suarez,' *Arch. de Méd. et de Pharm. Mil.*, vol. xxxvi. (1901), p. 470.
- SEGARD : 'Beri-beri in Madagascar,' *Arch. de Méd. Nav. Col.*, vol. xlv., p. 32.
- SEGUIN : Ship Cases, *Philadelphia Medical News*, December, 1886.
- SEGUIN and MASSEY : *Philadelphia Medical and Surgical Report*, 1888.
- SESTINI : 'Il beri-beri secondo le piu recenti ricerche etiologiche et anatomo-patologiche,' *Ann. di Med. Nav.*, September and October, 1898.
- SHATTUCK, F. C. : 'A Curious Epidemic' (on board the *Nelly Swift*), *Boston Medical and Surgical Journal*, vol. cv. (1881), pp. 400 and 577.
- SHEEN, A. : 'The Etiology of Peripheral Neuritis,' *British Medical Journal* (1901), i., p. 1377.
- SILVA LIMA, J. F. DA : 'Essaio sobre o beriberi no Brazil,' Bahia, 1872; abstract in *Arch. de Méd. Nav.*, vol. xx. (1873), p. 321.
- SIMMONS : 'Beri-beri, or the Kakké of Japan,' *Medical Report of the Imperial Maritime Customs*, China, 1880.
- SIMON, M. F. : 'Beri-beri in Singapore Gaol,' *Straits Settlements Government Gazette* (1883), p. 222.
- SIMON, MAX F. : (1) 'The Causes of Death in Beri-beri,' *Lancet*, March 8, 1893; (2) 'The Known and the Unknown in Respect of Beri-beri,' *Journal of Tropical Medicine*, September, 1899, p. 29; (3) 'The Causation of Beri-beri,' *ibid.*, September 2, 1901, p. 285.
- SLATER, W., and OLIVER, T. : 'Notes on an Outbreak of Beri-beri on the Chinese Transport Ship *Too Nan* stationed at Newcastle-upon-Tyne,' *Lancet* (1887), ii., p. 1125.
- SLOT : *Gen. Tijd. v. de Zeem.*, vol. ix., p. 312.
- SMART : *Report of the Surgeon-General U.S.A. Army*, 1902-1903, p. 69 ('Beri-beri among Filipinos').
- SMITH, J. H. : *Report of the Royal Commission on Arsenical Poisoning*, 1903 ('Sources of Glucose; Glucose in Food').
- SMITH, E. C. M. : *British Medical Journal* (1896), ii., p. 1791 ('Beri-beri on Ship *Lodestar*'); *British Medical Journal* (1898), ii., p. 1427 (recurrence on same ship, re-named *Steinbek*).
- SMYTH, J. : *British Medical Journal* (1889), i., p. 193.
- SODRÉ, A. DE AZEVEDO : 'Beri-beri,' *Twentieth Century Practice*, vol. xiv.

534 THE CAUSE AND PREVENTION OF BERI-BERI

- SPENCER, M. H. : ' Notes on Beri-beri as observed at the Seamen's Hospital, Greenwich,' *Lancet* (1897), i., p. 30.
- SPLEIDT, W. : ' Eine beri-beri epidemie au bord,' *Arch. f. Schiffs und Tropen Hyg.*, vol. iii. (1899), p. 207.
- SPRINGTHORPE : *Australasian Medical Gazette*, 1889.
- STANLEY, A. : ' The Nature of Beri-beri: An Etiological Study among Chinese Prisoners at Shanghai,' *Journal of Hygiene*, vol. ii. (1902), p. 369.
- STÉKOULIS : ' Une petite épidémie de béri-béri à bord d'une garde-côtes à Camaran,' *Janus*, vol. iv. (1899), p. 43.
- STRACHAN, H. : ' On a Form of Multiple Neuritis Prevalent in the West Indies,' *Practitioner*, vol. vi. (1897), p. 477.
- SWAVING : ' Beri-beri ' (at Batavia), *Geneesk. Tijdschr. v. Ned.-Ind.*, vol. xiv. (1874), p. 83.
- TABURET : ' Un cas de béri-béri suraigu,' *Journ. de Méd. de Bordeaux*, vol. xxxiii. (1903), p. 287.
- TAKAKI, K. : ' Prevention of Kakké in Japanese Navy,' *Sei-i-Kwai*, August, 1885 ; April, 1886 ; April and May, 1887.
- TAMSON, J. A. : ' Bijdrage tot de contagiositeit van beri-beri,' *Geneesk. Tijdschr. v. Ned.-Ind.*, vol. xxxvi. (1896), p. 88.
- TATTERSALL, C. H. : ' Report of County Medical Officer of Health to the Royal Commission on Arsenical Poisoning, 1900.'
- TATTERSALL, F. H., and DELÉPINE, S. : ' Special Report on the Epidemic of Arsenical Poisoning from Beer in 1900,' Salford, 1901.
- TAYLOR, F. : ' On Multiple Neuritis,' *Guy's Hospital Reports*, 1888.
- TAYLOR, W. : ' Studies in Japanese Kakké,' *Sei-i-Kwai*, August, 1885 ; May, 1886.
- THOMSON, W. : *British Medical Journal* (1898), i., p. 119 (' Beri-beri in South America ').
- TIBERIO, V. : ' Alcuni casi di beri-beri osservati sulla R.N. *Voltorno a Zanzibar*,' *Ann. di Med. Nav.*, vol. i. (1903), p. 705.
- TRAVERS, E. A. O. : (1) ' The Theory of the Causation of Beri-beri by a Toxin conveyed by Rice considered in the Light of Local Experience of the Disease,' *Journal of Tropical Medicine*, vol. v. (1902), p. 231. (2) ' Further Observations on the Rice Theory of Beri-beri, being a Reply to Dr. Braddon's Criticisms of Certain Pudooh Gaol Experiments,' *Selangor Government Press*, 1902. (3) ' Annual Report on the Medical Department,' *Selangor Government Gazette*, 1902. (4) ' Some Observations on Beri-beri,' *Journal of Tropical Medicine*, vol. vii. (1904), p. 285.
- TUCZEK, F. : ' Klin. und anat. Stud. über die Pellagra,' Berlin, 1893.
- TUNZELMANN, E. W. VON : ' A Contribution to the Study of Beri-beri,' *Lancet* (1894), ii., p. 1467.
- TURNER, H. G. : *St. Thomas's Hospital Reports*, vol. xxv., 1887 (' Peripheral Neuritis ').
- UCHERMANN, V. : (1) ' Laegebok fer Sjemoend,' 1895 ; (2) ' Ist beri-beri ein einheitliches krankheitsbild ?' *Zbl. f. innere mediz.*, xxv., 1904, p. 617 ; (3) with Abrahamson, Lars, and Kreyberg, P., ' Indstilling fra den af Dept fra det Indre . . . Kemite . . . of sygdemmen beri-beri on bord Norske skibe . . . etc.' (Report of Norwegian Committee on Beri-beri, Christiania, 1902).

- VILETTE : 'Contribution à l'étude de béri-béri,' *Thèse*, Lille, 1886.
- VINES, C. S. : 'Beri-beri in the Port of London,' *British Medical Journal* (1894), ii., p. 845.
- VOGL, A. F. : 'The Seed Fungus of Lolium,' *Zeitschr. f. Nahrungs Mitt. unters. Hyg. v. Waarenkind*, vol. ii. (1898), p. 28.
- VOORTHUIS, J. A. : 'Mededeeling over Beri-beri,' *Nederl. Tijdschr. v. Geneesk.* (1898), p. 41.
- VORDERMAN, A. G. : (1) 'Onderzoek naar het verband tusschen den aard der rijstroeding in de gevangenissen op Java en Madoera en het voorkomen van beri-beri onder de geïnterneerden,' Batavia, 1897. (2) 'Toelichting op mijn beri-beri verslag,' *Geneesk. Tijdschr. v. Ned.-Ind.*, vol. xxxviii. (1898), p. 47.
- WANKLYN, J. A. : 'Arsenic,' London, 1901.
- WETHERELL, J. A. : 'Beri-beri' (in Australian natives in prison), *British Medical Journal* (1894), ii., p. 950.
- WEINTRAUB, K. : 'Ueber Beri-beri,' *Wien. Med. Woch.*, 1887, No. 29, p. 964; 1888, No. 23, pp. 30, 44.
- WENDLAND : 'Ueber das auftreten der Beri-beri krankheit in Kaiser-Wilhelm's Land,' *Arch. f. Schiffs und Tropen Hyg.*, vol. i. (1897), p. 237.
- WERNICH, A. : (1) 'Klinische Untersuchungen über die Japanische Varietät der Beri-beri Krankheit,' *Virchow's Archiv*, vol. lxxi. (1877), p. 290. (2) 'Ueber die Beziehungen zwischen perniciöser Anämie, und Beriberikrankheit,' *Deut. Arch. f. Klin. Med.*, vol. xxi. (1878), p. 168.
- WERTENBAKEN : *U.S. Marine Hospitals Report*, 1895 (Ship Cases).
- WOODYATT, J. P. : 'Arsenical Beer Poisoning at the Halifax Union Poor-Law Hospital,' *British Medical Journal* (1902), i., p. 245.
- WORONIN : 'Ueber das "Tamelgetreide" in Sud-Ussurien,' *Botan. Zeit.*, vol. xlix. (1891), February 6; *Ext. Ann. Agronomiques*, t. xvii., 1891.
- WRIGHT, H. : (1) 'Changes in the Neuronal Centres in Beri-beric Neuritis,' *British Medical Journal* (1901), i., p. 1610. (2) 'An Inquiry into the Etiology and Pathology of Beri-beri. Studies from the Institute of Medical Research, Federated Malay States,' vol. ii., No. 1, Singapore, 1902. (3) 'On the Classification and Pathology of Beri-beri,' vol. ii., No. 2, London, 1903. (4) 'Beri-beri in Monkeys,' *Brain*, f. 2, 1903-04.
- YABÉ, TATSUSABURO : 'Disparition du Kakké dans la Marine Japonaise,' *Arch. de Méd. Nav.*, vol. lxxiii. (1900), p. 48.
- YAMAGIWA, K. : 'Beiträge zur Kenntniss der Kakké,' *Virch. Arch.*, bd. 156 (1899).

INDEX

The numbers refer to the pages.

- ABBEVILLE (Louisiana, U.S.A.), beri-beri in, 113
- Adulteration of wheaten flour, 441
- Ainos, beri-beri amongst, 419
- Alabama, Bryce Insane Hospital (U.S.A.), epidemic in, 430
- Alcoholic poisoning, close resemblance of, to beri-beri, 12, 18, 474
- Alcoholics, tendency of, to diseases generally, 450
- American troops in the Philippines, beri-beri amongst, 103, 224
- Ampanga, epidemic of beri-beri in, 186
- Ancona, epidemic on board of, 41, 62, 97 *et seq.*, 294
- Anderson (Major) on beri-beri in the ships of the Indian Marine Survey, 41, 62, 93, 294
- Androcles, beri-beri on board of, 99
- Annamese in New Caledonia, epidemic amongst, 234
- Annandale (Mr. Nelson), experiences in Upper Perak and Siam, 176
- Archer (Dr.) on an epidemic on board ship, 261
- Arkansas State Asylum, epidemic of beri-beri in, 434
- Arrowroot, adulteration of, 99, 443
- Arsenic in wall-papers, 32
- in glucose, 473
- large amount of, occasionally in beer, 473
- Arsenic-poisoning, close resemblance of, to beri-beri, 12, 18, 474
- Commission on, quoted, 63, 475
- probably not the cause of the 1900 epidemic in Lancashire, 476
- toxic effect of minute doses, 473
- Ashmead, objection to author's rice theory, 416
- Assam, Gaupati Gaol, beri-beri in, 87
- Asylums, special 'foci' of beri-beri, 449
- Atropine, effects of, resemble those of beri-beri poison, 385
- Australia, Chinese in, beri-beri amongst, 112
- natives, beri-beri amongst, 96
- Author's experiments in five Straits Settlements hospitals, 312 *et seq.*
- Average latent period of beri-beri, 86 *et seq.*
- Back on beri-beri in Japan, 36
- Baelz (Dr. B.) on beri-beri amongst Japanese troops, 231
- objections to author's rice theory, 416
- Bailey (Dr. J. W.) on an epidemic of beri-beri in Vancouver Island, 113
- Ballam rice non-productive of beri-beri, 112, 255
- Bangkok, outbreak of beri-beri in, 207
- Barner (Dr. W. B.), report on the Arkansas State Asylum epidemic, 434
- Barry (Dr. C. C.) on beri-beri in Rangoon, 36
- attributes it to excessive indulgence in rice-water, 211
- Batavia, beri-beri in, 262
- Beer largely manufactured from glucose, 446
- Beer-drinkers' epidemic of 1900 probably due to beri-beri, 476 *et seq.*
- Bengal Sepoy, diet of, 92, 267
- Bengalis, as a class, not rice-eaters, 126
- Bentong, beri-beri in, 221 *et seq.*
- Beri-beri, abrupt appearance and disappearance of epidemics, 53
- close resemblance of arsenic-poisoning to, 12, 18, 474
- muscarine-poisoning to, 384
- ptomaine-poisoning to, 378
- comparatively new disease in British Malaya, 175
- description of, 5 *et seq.*, 382 *et seq.*, 454 *et seq.*
- enormous mortality due to, 2
- extension of, owing to wider use of cheap milled rice, 206, 415

- Beri-beri, intractable to drugs, 9
 no name for, in Chinese, Burmese, or Siamese vernacular, 206
 not dependent on climate for its causation, 326
 not due to bacteria in the blood, 24 *et seq.*
 not due to local miasma, 33
 not infectious, 26 *et seq.*, 345
 occurrence of, in temperate climates doubtful, 426
 points of resemblance to Landry's paralysis, 11
 puerperal, 277
 surgical, 275
 uncertainty of diagnosis, 7, 162, 197, 475
- Beri-beric poison probably a stable non-volatile alkaloid, 385
 of complex and variable composition, 399
- Bidie* (Dr. G) on beri-beri in Madagascar, 210 (*note*)
- Blights upon grain, rapid spread of, 348
- Blyth* (Wynter) on flour adulteration, 442
- Boeck, Van*, on ergotism symptoms, 17
- Boer prisoners in St. Helena, epidemic amongst, 436
 diet of, 439
- Bolton* (Dr. W. G.) on an epidemic in Diego Garcia, 20
- Bondurant* (E. M.) on an epidemic in Tuscaloosa Asylum, 430 *et seq.*
 divergence of above from tropical type of beri-beri, 433
- Bourguignon* on beri-beri in Congo State, 272
- Boyé* (Dr.) on epidemics in French Guinea, 260
- Brazil, beri-beri in, follows importation of rice, 415
- Brémaud* on an epidemic on s.s. *Ilione*, 36
 on an epidemic in Poulo Condore, 258
 regards causation of beri-beri as dietetic, 39, 44
- 'Brewing-sugar,' a name for glucose, 445
 extensive use of, 445
 possible vehicle of beri-beric poison, 447
- British Guiana, beri-beri in, 415
- British Malaya, Bengalis in, exempt from beri-beri, 130
 Chinese immigrants form great majority of beri-beri patients in, 214
 conditions of contract labour in, 217
 diet of, 215, 218
 Europeans in, exempt from beri-beri, 129
 free coolies in, least liable to beri-beri, 221
- British sailors, exemption from beri-beri, due to absence of rice in rations, 245
 troops in Burma, beri-beri amongst, 107 (*note*)
- Brunton* (Dr. T. L.) on muscarine-poisoning, 380
- Bryson* (Captain R.) on beri-beri at Vizianagram, 269
- Buchanan* (Major W. J.) on preparation of rice, 147
 on beri-beri in India, 211
- Bullmore* (Dr. C.) on ship beri-beri, 63, 467 *et seq.*
- Burg* (C. L. van der) on beri-beri in the Dutch Army, 247
- Burma, British troops in, beri-beri amongst, 107 (*note*)
- Burmese gaols, beri-beri in, 86
- Bury* (Dr. J.) on peripheral neuritis, 424
- Byrne* (Surgeon-General) on beri-beri amongst American troops in the Philippines, 409
- Calcutta, Chinese in, beri-beri amongst, 114
- Calverly* (Dr. C. S.), report of epidemic in Rutland County, Vermont (U.S.A.), 435
- Campbell* on beri-beri in Gaupati Gaol, Assam, 87
- Casey* (Dr. J. P. N.), description of epidemic amongst Boer prisoners in St. Helena, 436
- Cereals, properties common to, 43
 all liable to beri-beric toxification, 399, 448
- Change of food and surroundings, therapeutic value of, in beri-beri, 9
- Chantabun, epidemic in, 258
- Chapman* (Mr. W. T.) on beri-beri on Christmas Island, 317
- China comparatively free from beri-beri, 44
- Chinese cheap rice a source of beri-beri, 232
 immigrants in British Malaya most liable to beri-beri, 36, 214
 diet of ditto, 215-218
- Christmas Island, beri-beri on, 64, 173, 314 *et seq.*, 421
- Claviceps purpurea*, 46
- Climate no factor in the causation of beri-beri, 326
- Commission on Arsenic-poisoning, 63, 475
- Comparative analysis of rice with other cereals, 139
 toxicity of different rices, determination of, 309
- Comparison of various diets in England and Japan, 228
- Conclusions from hospital statistics, 169
- Conditions under which rice becomes toxic, 370 *et seq.*
 hygienic. See Hygienic conditions

- 'Cones' flour, 442
 Conflicting features of beri-beri explained by author's rice theory, 321 *et seq.*
 Congo State, beri-beri in, 272
 Contagion, beri-beri not spread by, 53
 Corlette (Dr. C.) on beri-beri amongst Chinese in Australia, 114
 Corney (Dr. G.) on beri-beri amongst Japanese in Fiji, 112, 255
 Cossack pearling fleet, beri-beri in, 62, 94
 Curot on the causation of beri-beri, 30
 Daniels (Dr. C. W.) on beri-beri in Straits Settlements gaols, 300
 Darnel, special fungus on, analogous to rice-fungus, 393
 Davidson (Dr. A.), objection to author's rice theory, 42
 on beri-beri amongst the troops in the Dutch East Indies, 106
 Delépine (Dr. S.) on glucose in beer, 473
 Description of beri-beri, 5, 6, 382 *et seq.*, 454 *et seq.*
 Determination of comparative toxicity of different rices, 309
 Diagnosis, uncertainty of, in beri-beri, 7, 162, 197, 475
 Diego Garcia, epidemic in, 29
 Diego Suarez, epidemics in, 198 *et seq.*, 295
 Dieren (E. van), criticisms of Vorderman's results, 205 *et seq.*
 on beri-beri in Dutch Navy, 246, 281
 on toxic rice as a cause of beri-beri, 41
 Diet as a factor in the etiology of beri-beri, 39 *et seq.*
 of Boer prisoners in St. Helena, 439
 of European crews in Dutch East India Navy, 241
 of Indian native troops, 116, 267, 270
 in Java gaols, 265
 of Lascars on British ships, 451
 in Malay and Straits Settlements gaols, 68
 on Norwegian ships, 468
 Diets, comparison of, in England and Japan, 228
 Dropsy often mistaken for beri-beri, 197, 414
 Drugs therapeutically useless in beri-beri, 9
 Dryepondt on beri-beri in the Congo State, 272
 Dublin, Richmond Asylum, epidemics in, 86, 426 *et seq.*
 diet of inmates, 430
 disease clinically divergent from tropical type of beri-beri, 429
 Durham (Dr. H. E.), on beri-beri on Christmas Island, 7, 314
 on beri-beric symptoms, 10, 28 *et seq.*
 on causation of beri-beri, 217
 objections to author's rice theory, 421
 Dutch East Indian Army, beri-beri in, 105, 196, 246 *et seq.*
 Navy, beri-beri in, 240 *et seq.*, 281
 dietary of, 241
 Eecke (Van) on nerve-lesions in beri-beri, 14
 Eijkman (C.), experiments on fowls, 41, 357
 Ellis (Dr. W. G.), dietetic experiment in Singapore Lunatic Asylum, 405
 conclusions probably based on incorrect diagnosis, 407
 does not disprove author's rice theory, 406
 mistakes epidemic œdema for beri-beri, 414
 on race-incidence of beri-beri, 119
 report of beri-beri in Singapore Lunatic Asylum, 304
 on results of autopsies in cases of beri-beri, 15, 18
 Endemicity, true, not shown by beri-beri, 53
 Enzymes, action of, 386 *et seq.*
 Epidemic œdema, description of, 19, 408
 differentiated from beri-beri, 407
 Epidemics of beri-beri, abrupt appearance and disappearance of, 53
 seasonal and secular variations in, 53, 342
 Epileptics, liability of, to beri-beri, 449
 Ergotism, 17, 46, 257
 European troops in Dutch East Indies, beri-beri amongst, 105
 Europeans generally exempt where beri-beri is rife, 106, 119, 124, 129
 Experiments, author's, on different rices in Straits Settlements hospitals, 312 *et seq.*
 on animals, 354 *et seq.*
 Eijkman's, on fowls, 41, 357
 Fry's, on inmates of Penang Pauper Hospital, 310
 Grijns', on fowls, 358
 Hose's, on monkeys, 309
 Sakaki's, on fowls, 358
 Vorderman's, on inmates of Java gaols, 200
 Wright's, on monkeys, 355
 Fat, effect of, as a component of diet in beri-beri, 258 *et seq.*
 Ferments, action of, on food-stuffs, 48 (note), 386 *et seq.*

- Féris* on beri-beri in Brazil, 36, 415
Fiebig (M.) on beri-beri amongst natives of Malay Archipelago, 196
 objection to author's rice theory, 418
Fiji, Japanese coolies in, beri-beri amongst, 61, 111, 254, 256
Firket on beri-beri in Congo State, 272 *et seq.*
 report on beri-beri amongst Japanese in New Caledonia, 238
Fish, bad, how far responsible for beri-beri, 40
Flour, adulteration of, 442
Foussagrives, report of beri-beri on the *Parmentier*, 297
Forte (H.M.S.), beri-beri amongst Kroomen on, 245
Fowls, experiments on, 41, 357 *et seq.*
 polyneuritis in, resembles beri-beri, 356, 359
 induced by feeding solely on 'white' rice, 357
Fox on infrequency of beri-beri amongst Malays, 176
Free coolies in British Malaya least liable to beri-beri, 221
French Guinea, epidemics in, 260
Fry (Dr. W. S.), experiment in Penang Pauper Hospital, 310
Fungi in rye and darnel analogous to rice-fungus, 392 *et seq.*
- Gelphe* on fish diet as causative of beri-beri, 40
Gervard (Dr. P. N.) on latent period of beri-beri, 87
Gimlette (Dr. J. D.) on beri-beri amongst Sinkhehs at Bentong, 221
Glucose a possible cause of beri-beri, 444
 arsenic in, 473
 from what materials made, 443
 how manufactured, 444
 large use of, in manufacturing beer, 445
 in adulterating treacle, 446
Gowers (Dr. W. R.) on causes of multiple peripheral neuritis, 424
Grains, different, liable to infection by beri-beric agent, 346
Grall on diet as causative of beri-beri, 40
Grijns, experiments on fowls, 358
Grimm (F.) on beri-beri in Yezo, 107
- Hagen* on the supposed contagiousness of beri-beri, 110, 235
Hahn on the action of enzymes, 386
Hartley (Dr. W. D.) on the epidemic amongst Boer prisoners in St. Helena, 322
Harvest (rice), relation to beri-beri, 327, 347
Hassall on adulteration of arrowroot, 99 on 'cones' flour, 442
- Haynes* on beri-beri in the Cossack pearling fleet, 62, 94 *et seq.*
 on rice as causative of beri-beri, 95
Heret on adulteration of flour, 442
Hight (Dr. J. Campbell) on beri-beri in Siam, 271
Hindu care in the preparation of food, 126
Hirsch on the latent period of beri-beri, 64
 on physique as determining incidence of beri-beri, 323 (*note*)
Hoffman on the latent period of beri-beri, 63
Horses, beri-beri in, 350
 beri-beri in, due to feeding on 'padi,' 354
 how fed in the East, 353
 paralysis of, in temperate climates, 356
Hose (Dr. C.), experiments in Borneo, 307 *et seq.*, 355
 on beri-beri amongst Dyaks, 184
 on rice as causative of beri-beri, 299 *et seq.*
Hospitals (Straits Settlements), inferior rice used in, 285
Hygienic conditions (so called) have no influence in determining beri-beri, 274
- Immunity* against diseases generally, how acquired, 331 *et seq.*
 not produced by beri-beri against fresh attacks, 55
Imported rice generally the cause of beri-beri, 322, 341
Incidence of beri-beri, specially on the well fed, 256
 usually less on females than males, 256
 peculiarly severe on the insane, 449
India, beri-beri comparatively rare in, 210
Indian gaols, careful preparation of rice rations in, 210
 Marine Survey, beri-beri in ships of, 93 *et seq.*, 294
 Native Army, beri-beri in, 115
 troops, daily ration of, 116, 267
Infantile poliomyelitis generally allied with beri-beri, 12
Infection theory of beri-beri disproved, 26 *et seq.*, 345
Influenza, transportability of, 33, 345
Insane, the, beri-beri especially prevalent amongst, 449
- Japan*, beri-beri always present in, 108
 chiefly in summer, 232
 greater incidence of beri-beri upon the well-to-do, 417
Japanese Army, beri-beri in, 230 *et seq.*

- Japanese coolies in Fiji, beri-beri in, 61, 111, 254, 256
 in New Caledonia, beri-beri in, 237 *et seq.*
 Fleet, beri-beri in, 224 *et seq.*
 training-ships, beri-beri in, 226
 Japara District Prison, beri-beri, in 298
 Java gaols, Vorderman's investigations in, 200 *et seq.*
 dietary of, 265
 Joynt (H. N.) on beri-beri amongst Japanese coolies in Fiji, 61
 Judet de la Combe on beri-beri amongst Japanese coolies in New Caledonia, 110, 238
- Kaiser Wilhelm's Land, Europeans in, exempt from beri-beri, 107
 Kermogant (R.) on mortality amongst French convicts in New Caledonia, 109
 Kirchberg (E.) on three cases of beri-beri amongst Norwegian sailors, 464 (note)
 Kirk (Dr. J.) on exemption of Europeans from beri-beri in Singapore-Gaol, 123
 Königer on an epidemy of beri-beri in Manila, 100 *et seq.*, 223
 Korea, beri-beri amongst Japanese in, 113
 Kuala Lumpur, beri-beri amongst Malay police in, 185
 Gaol, beri-beri in, 216
 'Kumri,' a paralytic disease amongst horses, 351
- Lacerda (J. B. de) on a pestilence amongst animals in Island of Marajo, 350
 Laler (Captain N. P. O'Gorman) on beri-beri amongst Madras Infantry in Rangoon, 270
 Landry's paralysis, how far beri-beri resembles, 11
 Lascars in P. and O. vessel, beri-beri amongst, 96
 Lasnet on beri-beri in Dakar (Senegal) Prison, 86, 295
 Latent period in acute infectious diseases, 58
 in beri-beri extremely irregular, 53, 59
 minimum, 60
 importance of, in etiology of beri-beri, 206
 Lathyrism distinguished from beri-beri, 353
 'Laukheh,' definition of, 218
 Laurent (L.) on the dietetic effect of fat in beri-beri, 257 *et seq.*
 Leash, report on beri-beri in Singapore Prison, 73 *et seq.*
 Leapingwell (Surgeon - Lieutenant - Colonel) on beri-beri amongst Madras Infantry in Vizianagram, 268
- Leent (van) on beri-beri associated with scorbutus, 20 *et seq.*
 on beri-beri in the Dutch East Indian Navy, 240 *et seq.*, 280 *et seq.*
 on dietetic origin of beri-beri, 39
 Legrand on diet and infection as causes of beri-beri, 29
 Leroy de Mericourt on beri-beri on the *Parmentier*, 297
 Leslie (Dr. T. T.) on epidemics of beri-beri in Burma gaols, 86, 211
 Littlefield on the effects of fresh and stale rice in Philippine gaols, 295
 Locality, apparent influence of, on beri-beri, how explained, 52
 change of, incorrectly regarded as conditioning beri-beri, 280 *et seq.*
 not endemic for beri-beri, 324
 Lucy (Dr. S. H. R.) on beri-beri amongst Chinese coolies in Sungei Lembing mines, 329
 on beri-beri in the Pudooh Gaol, 171
 in the Penang Gaol, 301
 confirms author's theory, 302 (note)
- MacDougall (Dr.), statistics of beri-beri at Christmas Island, 315
 MacLeod (Dr. K.) on an epidemy of beri-beri in Calcutta, 19 *et seq.*
 on beri-beri on the *Ancona*, 62, 97 *et seq.*
 on diet as causative of beri-beri, 41
 MacMullen (Dr.) on beri-beri on the *Androcles*, 99
 Madagascar, beri-beri amongst troops in, 198, 365
 epidemics in, 260
 Madras troops especially liable to beri-beri, 115, 210, 268
 Magelang Military Hospital, beri-beri in, 263
 Maitland (Dr. C. B.) on causes of peripheral neuritis, 424
 Malacca Training College, beri-beri in, 181 *et seq.*, 295
 Malay Peninsula, beri-beri in, 64 *et seq.*
 Malays exempt from beri-beri in their primitive condition, 179, 190 *et seq.*
 liable to it abroad, 179
 susceptibility to beri-beri, 177 *et seq.*
 Malcohmmer on diet as influencing beri-beri, 92
 Malcolmson on diet as influencing beri-beri, 115
 Malt substitutes, growing use of in England, 445
 prohibited in Germany, 445
 probable source of beri-beri, 447

- Manila, beri-beri in, 36, 100 *et seq.*, 223
Manson (Dr. P.), definition of beri-beri, 5, 39, 454
 Miasmatic origin of beri-beri disproved, 33
Mills (Dr.) on beri-beri in Shanghai Municipal Gaol, 7
 Minimum latent period in beri-beri, 60
 temperature at which enzymes become inactive, 386
Miura (M.) on fish diet as causative of beri-beri, 40, 262
 Monkeys, experiments on, 309, 355
 Mortality due to beri-beri, 2
Mosse (Lieutenant-Colonel C. G. D.) on beri-beri amongst Boer prisoners in St. Helena, 438 *et seq.*
Mugliston (Dr. T. C.), report on experimental feeding of patients in Penang Pauper Hospital, 311 (*note*)
 Multiple peripheral neuritis, 344
 epidemy of, in and round Manchester, 345
 various causes of, 424
 Muscarine-poisoning, 380
 close analogy with beri-beri, 384
Nancowry and *Investigator*, beri-beri on, 60 *et seq.*
 Negri Sembilan, beri-beri amongst troops in, 185
 Negro troops in Madagascar, beri-beri amongst, 105
 New Caledonia, epidemics in, 40, 109, 234, 295
Nightingale (Dr.) on beri-beri in Bangkok, 207 *et seq.*
 Nijgata Prison, outbreak of beri-beri in, 232
 Nitrogen, deficiency of, in food no direct cause of beri-beri, 227
Nocht, objections to author's rice theory, 465
Norman (Dr. C.) on the epidemic in Dublin Asylum, 86, 464 (*note*)
 Norwegian Commission on beri-beri, 457, 465
 Reports of Captains, 457 *et seq.*; of English physicians, 459 *et seq.*
 ships, diet on, 468
 Nosology of beri-beri, 5 *et seq.*, 10 *et seq.*, 382 *et seq.*, 454 *et seq.*
 Œdema, epidemic, often confounded with beri-beri, 237, 407
 Okinawa (Loochoo Islands), beri-beri amongst Japanese in, 113
Oppenheim (Dr. H.) on peripheral neuritis, 28, 424
O'Reilly on beri-beri amongst American troops in the Philippines, 104
 Organic poisons, origin of, 386
Orr on cases of beri-beri on a P. and O. vessel, 96
Overbeck de Meijer on beri-beri in the Dutch East Indies, 106
 Paõre Mine, epidemy amongst Malay labourers in, 179 *et seq.*
Parmentier, beri-beri on, 297
 Parturition, especial incidence of beri-beri after, 277 *et seq.*
 Pearlina fleet (Cossack), beri-beri on, 62 *et seq.*, 94 *et seq.*, 294
Pekelharing (Dr. C. A.) on latent period of beri-beri, 63
 Pellagra, 47, 441
 Penang Lock Hospital, statistics of, 168
 Pauper Hospital, Dr. W. S. Fry's experiment in, 310
 Prison, beri-beri in, 301
 Rural Tamils in, less liable to beri-beri than urban, 167, 171, 187
 Perak Hospital, statistics of, 156 *et seq.*
 Period of incubation in acute infectious diseases, 58
 in beri-beri extremely irregular, 59
 average duration, 86 *et seq.*
 minimum, 60
 Periodicity of beri-beri outbreaks, conditions which determine, 317 *et seq.*
 synchronous in different localities, 339
 Peripheral neuritis. See Multiple Peripheral Neuritis
 Personal conditions modify the effect of the beri-beri poison, 275
Petit on beri-beri amongst Senegalese troops in Madagascar, 198, 260, 365
 on diet as causative of beri-beri, 41
 Philippines, beri-beri amongst American troops in, 103, 409
 Physical properties of beri-beri poison in rice, 377
 Physique as a determining factor in beri-beri, 323 (*note*)
 Pig, rice-fed, paralysis in, 355
Pinard (Dr. E.) on beri-beri amongst Senegalese troops in French Guinea, 261
 Poliomyelitis, infantile, generally allied with beri-beri, 12
 Politico-economical effects of beri-beri, 5
Porée on beri-beri epidemics amongst coolies in New Caledonia, 40
 Port Dickson, epidemy of beri-beri in, 188
 Pulo-Condore, epidemics of beri-beri in, 285 *et seq.*
 Prison diet in Java, 265
 in Straits Settlements and Federated Malay States, 68
 Prisoners' length of sentence as determining incidence of beri-beri, 290, 310 (*note*)

- 'Privatory' theory of origin of beri-beri, 368
disproved, 370
- Proofs of author's rice theory summarized, 319
- Ptomaine-poisoning most closely resembles beri-beri, 378
- pathology of, 379
- on board ships, 466
- Public institutions peculiarly liable to beri-beri, 54, 449
- Pudoh Gaol, account of beri-beri in, 80 *et seq.*, 172
- Travers' experiment in, 287, 481 *et seq.*
- Wright's experiment in, 287 *et seq.*
- Puerperal beri-beri, 277
- Quill (Lieutenant-Colonel R. A.) on beri-beri amongst Indian troops at Trincomalee, 270
- Rainfall in British Malaya, 327
bears no definite relation to beri-beri, 335 *et seq.*
- Randall (Dr. P. W.) on beri-beri in Ascension, 20
- Rangoon, troops in, beri-beri amongst, 115, 270
- Reaucar on surgical beri-beri, 276
on characteristic symptoms of beri-beri, 381 *et seq.*
- Recruits, special incidence of beri-beri on, in Japanese Army, 266
- 'Red' rice. See Rice
- Rees (Dr. D. C.) on ships as infected centres of beri-beri, 34 *et seq.*
- Religion differentiates the victims of beri-beri, 274
- Rice a staple food in beri-beric countries, 44
age of, after harvest, determines development of beri-beri, 328, 364
beri-beric agent a surface parasite *on*, not *in*, the seed, 391
insoluble in cold water, and non-volatile, 377
not endogenic in fresh seeds, but adventitious, 371
peculiar to this grain, 371
probably not due to a ferment, 389
but to a special fungus, itself toxic, 390
resists high temperatures, 377
conditions governing toxicity of, 49 *et seq.*
- 'cured,' 149 *et seq.*
description of, 147
preparation of, in India, 146 *et seq.*
- Rice, 'cured,' preparation of, destroys the toxin-forming agent, 376
results in the most nutritious product, 147, 374
decortication of, followed by, but not the cause of, beri-beri, 372 *et seq.*
entails devitalization of grain, 373
devitalized grain powerless to resist infection, 374
dietetic value of, depends on treatment of raw grain, 140
'fresh,' used mostly in Java, 149
consumption of mainly confined to growers of it, 144
may occasionally be toxic, 398
immense number of varieties of, 136
imported, generally the cause of beri-beri, 322, 417
infectious nature of toxin in, 51
methods of eliminating toxic agent from, 50
mode of preparation of raw grain amongst Malays, 141; in Burma, China, and Dutch Indies, 144
mode of preparation of raw grain determines incidence of beri-beri, 51
most innutritious of all cereals, 140
not the *immediate* cause of beri-beri, 45
peculiarly liable to parasitic attacks, 46
pericarp of, contains little or no poison, 372
'red,' 141
does not convey beri-beri, 203, 372
but is no antidote against it, 204
more nutritious than 'white,' 139, 149
small proportion of fat in, 140
structure and composition of, 137 *et seq.*
toxic, confined to definite areas, 51
transport and storage of, as determining factors in beri-beri, 54
'uncured,' or 'stale white,' the rice of commerce, 144, 149
cheap Chinese source of beri-beri, 232
contains toxic beri-beric agent, 370
most expensive kinds of, least nutritious, 145
process of milling facilitates deterioration, 146
removes most valuable part of grain, 145
toxification of, explained, 398

- Rices, different, determination of comparative toxicity of, 309
- Rice-supply, relation of, to beri-beri, 91
- Rice-water an assumed cause of beri-beri, 211
- Richmond Asylum. See Dublin
- Riujo* (Japanese training-ship), beri-beri on, 298
- Robinson* (Mr. F.) on an epidemic of beri-beri in the Malacca Training College, 183 *et seq.*
- Rost* (Dr. E. R.) on rice-water as causative of beri-beri, 211
- Rowell* (Dr. T. J.) on an epidemic in Singapore Gaol, 88 *et seq.*, 120
- Rutland County (Vermont, U.S.A.), epidemic in, 435
- Ruxton* (Dr.) on ptomaine-poisoning, 381
- Rye, special fungus on, analogous to rice fungus, 392
- fructification of, 398
- Sago a possible cause of peripheral neuritis, 420
- Sailors, inferior diet of, 451
- Sakaki*, experiments on fowls, 358 *et seq.*
- author's criticisms on, 361 *et seq.*
- Saneyoshi* on diet as sole cause of beri-beri, 113, 224, 230, 234, 262
- on fish as non-causative of beri-beri, 41
- on local incidence of beri-beri, 107, 416
- statistics of beri-beri in Japanese Navy, 229
- Sausage-poisoning, 380
- Scheube* on latent period of beri-beri, 64
- objections to author's rice theory, 414, 416
- Schuttelaere*, account of two epidemics in Diego Suarez, 198
- on rice as sole cause of beri-beri, 295
- Seasonal variations in beri-beri independent of local conditions, 341
- synchronous in different stations, 339, 342
- Selangor Gaol, account of beri-beri in, 78 *et seq.*, 164, 171, 252
- dietary of, 511
- Dr. E. A. O. Travers' experiment in, 481 *et seq.*
- author's criticism on, 483 *et seq.*
- Senegal, Dakar Prison, beri-beri in, 86
- Senegalese troops in Madagascar, beri-beri amongst, 198
- Shanghai gaols, beri-beri in, 87
- Shimushu Island, history of beri-beri in, 234
- Ships, beri-beri on, 271
- miasmatic theory of, 93
- probably often incorrectly diagnosed, 453, 464
- Siam, beri-beri in, 271
- Simmons* (Dr. D.) on miasmatic theory of beri-beri, 318
- Sinclair* (Surgeon-Major-General) on 'beri-beri amongst Madras troops, 115
- Singapore Gaol, beri-beri in, 69 *et seq.*, 120, 172, 249
- variations of, with change of diet, 305 *et seq.*, 501 *et seq.*
- General Hospital, statistics of beri-beri in, 168, 186
- Lunatic Asylum, Dr. W. G. Ellis's experiment in, 302 *et seq.*
- excess of rice in diet of, 254
- statistics of beri-beri in, 118
- 'Sinkheh,' definition of, 217
- especially liable to beri-beri, 221
- Sheat* (Mr. W.), case of beri-beri, 185
- Smart* (Surgeon-General) on beri-beri amongst the Philippine Scouts, 103
- objection to author's rice theory, 408; subsequently modified, 409
- Smith* (Dr. Hammond) on glucose as an adulterant, 446
- Smyth* (Dr. J.) on beri-beri amongst Madras troops in the Soudan, 117
- Soudan, beri-beri amongst Madras troops in, 116
- South Africa, beri-beri in, 322
- Southern Japan, Europeans and Americans exempt from beri-beri in, 108
- Special incidence of beri-beri on the well fed, 256
- Spread of beri-beri owing to extending use of cheap milled rice, 206, 415
- Stanley* (Dr. A.) on the causation of beri-beri, 124
- objection to author's rice theory, 418
- Staple food-stuffs of South America, 415
- State prohibition of all 'uncured' rice will stop beri-beri, 479
- Statistics, comparative, of shipping according to nationality, 468
- of beri-beri on Christmas Island, 315
- in the Dutch Army in the East, 247 *et seq.*
- in hospitals throughout British Malaya, 194 *et seq.*
- among immigrant coolies in the Straits Settlements and Native States, 158 *et seq.*
- in the Japanese Navy, 229
- amongst Madras troops, 267
- in Penang Lock Hospital, 168

LANE MEDICAL LIBRARY

To avoid fine, this book should be returned on
or before the date last stamped below.

DEC 9 2004
DEC 19 2004
JAN 11 2005

JAN 13 2005

L122 Braddon, W.L. 43241
B79 The cause and preven-
1907 tion of beri-beri

NAME

DATE PAID

